SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER

A THESIS SUBMITTED TO THE BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT (PUNJAB)

FOR THE AWARD OF DEGREE OF

MD

IN

MEDICINE

BY

DR. ADITI MATHUR

SUPERVISOR

DR. SANDEEP CHHABRA

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA
(MAY, 2021)

DECLARATION

I declare that the thesis entitled **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** has been prepared by me under the guidance of **Dr. Sandeep Chhabra**, Associate Professor, Department of Medicine, Dayanand Medical College and Hospital, Ludhiana. No part of this thesis has formed the basis for the award of any degree previously.

Dr. Aditi Mathur
Department of Medicine
Dayanand Medical College & Hospital
Tagore Nagar
Ludhiana-141001
Punjab

Date:

CERTIFICATE

I certify that **Dr. Aditi Mathur** has prepared her thesis entitled **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** for the award of degree of M.D. in Medicine of Baba Farid University of Health Sciences, under my guidance. She has carried out the work at the Department of Medicine, Dayanand Medical College & Hospital, Ludhiana.

Supervisor

Dr. Sandeep Chhabra
Associate Professor
Department of Medicine
DMCH, Ludhiana

Co-Supervisor(s)

Dr. Narender Pal Jain
Professor
Department of Medicine
DMCH, Ludhiana

Dr. Bhupinder Singh
Associate Professor
Department of Cardiology
DMCH, Ludhiana

Date:

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT AND PRINCIPAL

This is to certify that the thesis entitled **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** is a bonafide research work done by **Dr. Aditi Mathur** under the guidance of **Dr. Sandeep Chhabra**, Associate Professor, Department of Medicine, Dayanand Medical College & Hospital, Ludhiana.

Head of Department

Dr. Dinesh Gupta
Professor & Head
Department of Medicine
DMCH, Ludhiana

Principal

Dr. Sandeep Puri
Principal
DMCH, Ludhiana

Date:

COPYRIGHT

Declaration by the Candidate

I hereby declare that the Baba Farid University of Health Sciences, Faridkot, shall have the rights to preserve, use and disseminate this thesis in print or electronic format for academic / research purpose.

Dr. Aditi Mathur
Department of Medicine
Dayanand Medical College & Hospital
Tagore Nagar
Ludhiana-141001
Punjab

Date:

© Baba Farid University of Health Sciences,

Faridkot

Dedicated

to

my family

&

teachers

ACKNOWLEDGEMENT

Right at the outset, I deem it de rigueur to dedicate this humble venture to the Almighty, the omnipotent, omnipresent, master of this whole universe who lead me along, holding me by the finger. Where ever I faltered along the way, He was always there to pick me up in His lap like an endearing parent to tide over the obstacles.

Endeavor, no matter how spirited and consistent does little to steer one, unless coupled with able guidance. With great pleasure I take this opportunity to thank all those who helped me in the successful completion of my work. Not to fulfill a formality, but to express the feeling in my heart, I gratefully acknowledge the keen personal interest and guidance rendered by my esteemed & revered teacher, mentor and Chief Supervisor **Dr. Sandeep Chhabra,** Associate Professor, Department of Medicine, Dayanand Medical College and Hospital, Ludhiana. His innovative ideas and accommodative attitude helped me at various stages during the culmination of this project. I consider myself privileged to be his student. I deem myself singularly blessed of being showered with parental love, guidance, constructive criticism and inspiration of my spirited teacher. This project could never have been brought to its culmination without his dedicated involvement, patient hearing and enthusiastic help at each and every step of this journey. His thorough knowledge of the subject and pursuit for perfection has been a constant inspiration. I wish to express my deep & heartful gratitude for my esteemed teacher & co-supervisor **Dr. Bhupinder Singh,** Associate Professor, Department of Cardiology, DMCH, Ludhiana. His constant kindness, courtesy, meticulous care, encouragement, untiring help and constructive suggestions helped me through- out my study. It was his contribution and dedicated involvement which gave me the courage to materialize this work. Words would be amiss without a mention of **Dr Narender Pal Jain,** Professor, Department of Medicine, DMCH, for his invaluable suggestions & opinions & constant support that helped tremendously in completion of this project.

I am also indebted to **Dr Sandeep Puri,** Principal, DMCH, Ludhiana for his guidance and untiring supervision. I cannot thank **Dr Dinesh Gupta**, Professor & Head, Department of Medicine, DMCH, enough for all the encouragement and guidance he has provided in the completion of this work. He has been a source of constant inspiration to me.

Parents are always a perpetual source of inspiration & encouragement. No words can express what their constant love, sacrifice, & prayers have helped me achieve. A very sincere thanks to my Dad Dr Manoj Mathur and Mom Dr Nidhi Mathur. My younger brother Kanav Mathur, who is presently pursuing MBBS in DMCH, has also been a great help in running odd errands for my thesis. Last but not the least I would like to thank Mrs Namita Bansal, Mr Naveen Bhatia, Jaswinder Kaur and all the R&D staff for their skillful analysis & typing of the manuscript. I acknowledge the help of all those invisible hands who are responsible for the successful outcome of this dissertation.

Dr. Aditi Mathur…

ABBREVIATIONS

1.	AF	Atrial Fibrillation
2.	ADE	Antibody Dependent Enhancement
3.	CPK-MB	Creatinine Phosphokinase MB
4.	CRCL	Creatinine Clearance
5.	DF	Dengue Fever
6.	DHF	Dengue Hemorrhagic Fever
7.	DSS	Dengue Shock Syndrome
8.	EDS	Expanded Dengue Syndrome
9.	ECG	Electrocardiogram
10.	ECHO	Echocardiography
11.	HR	Heart rate
12.	LVEF	Left Ventricular Ejection Fraction
13.	LDH	Lactate Dehydrogenase
14.	MRI	Magnetic Resonance Imaging
15.	PLT	Platelets
16.	RTPCR	Real Time Polymerase Chain Reaction
17.	TRT	Troponin-T
18.	WHO	World Health Organisation.

ABSTRACT

Introduction: Dengue is caused by an arbovirus belonging to the genus flavivirus, which is transmitted by the bite of female Aedes aegypti and Aedes albopictus mosquitos.

Aims and Objectives: 1.To study the spectrum of cardiac manifestations in dengue fever. 2.To ascertain the impact of cardiac abnormalities on patient outcome.

Material and Methods: This study was performed in the department of medicine, Dayanand Medical College and Hospital (DMCH) over a period of one year. All cases of dengue fever and those with cardiac complications were included in the study. In every case, a detailed clinical history and examination, with special attention to cardiac manifestations, was taken. Diagnosis of dengue fever was established by serological examination. Cardiac manifestations were established by ECG, echo & cardiac biomarkers evaluation.

Results: The present study was conducted in DMCH Ludhiana on 281 cases of dengue fever. The age distribution of patients was from 10 to 69 years with the mean age of 41.9 years. There were 200 male patients in the study (71.17%) and 81 female patients (28.83%).88.97% of patients showed thrombocytopenia. There was no significant co-relation between thrombocytopenia, leucopenia and cardiac manifestations. As regards co-morbid conditions, cardiac manifestations were more common in patients of chronic kidney disease, type II diabetes and hypertension. This co-relation was statistically significant with a p value of <0.001. The spectrum of cardiac manifestations in dengue included bundle branch block, bradycardia, ectopics, Myocardial injury, tachycardia, left ventricular hypertrophy and mitral regurgitation. Bradycardia was the most common cardiac manifestation.

Conclusion: Most of the cardiac manifestations were reversible and rhythm abnormalities, ectopics and bundle branch block were transient and self-limiting in nature. The myocardial injury was also found to be reversible in majority of the patients. The results were statistically highly significant with a p value of <0.001.

Key words: Bundle Branch Block, Ectopics, Tachycardia, Left Ventricular Hypertrophy Mitral Regurgitation, Bradycardia.

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-8
II	AIMS AND OBJECTIVES	9
III	REVIEW OF LITERATURE	10-30
IV	MATERIAL AND METHODS	31-32
V	OBSERVATIONS AND RESULTS	33-61
VI	DISCUSSION	62-67
VII	SUMMARY AND CONCLUSION	68-69
VIII	BIBLIOGRAPHY	70-74
	APPENDICES – I - PROFORMA - MASTER CHART	
	APPENDICES – II - APPROVAL LETTER OF THESIS PLAN - THESIS PLAN	

LIST OF TABLES

Table No.	Title	Page No.
1.	AGE DISTRIBUTION	33
2.	GENDER DISTRIBUTION	34
3.	TLC DISTRIBUTION	35
4.	DLC NEUTROPHILS DISTRIBUTION	36
5.	DLC LYMPHOCYTE DISTRIBUTION	37
6.	PLATELET COUNT DISTRIBUTION	38
7.	DISTRIBUTION OF CREATININE CLEARANCE (CRCL)	39
8.	SGOT DISTRIBUTION ACCORDING TO GENDER	40
9.	SGPT DISTRIBUTION ACCORDING TO GENDER	41
10.	RATIO OF SGOT/SGPT	42
11.	CHEST X-RAY (CXR) FINDINGS	43
12.	HEART RATE DURING HOSPITAL STAY	44
13.	ECTOPICS DISTRIBUTION	45
14.	BRADYCARDIA DISTRIBUTION	46
15.	TACHYCARDIA DISTRIBUTION	47
16.	LEFT VENTRICULAR EJECTION FRACTION (LVEF) DISTRIBUTION	48
17.	CREATINE KINASE-MB (CKMB) DISTRIBUTION	49
18.	TROPONIN-T (TRT) DISTRIBUTION	50
19.	D-DIMER DISTRIBUTION	51
20.	MYOGLOBIN DISTRIBUTION	52
21.	LACTATE DEHYDROGENASE (LDH) DISTRIBUTION	53
22.	CARDIAC MANIFESTATION DISTRIBUTION	54
23.	OUTCOME OF CARDIAC MANIFESTATIONS	56
24.	CO-RELATION BETWEEN TLC AND CARDIAC MANIFESTATIONS	58

25.	CO-RELATION BETWEEN PLATELET COUNT AND CARDIAC MANIFESTATIONS	59
26.	CO-RELATION BETWEEN CRITICAL PLATELET COUNT AND CARDIAC MANIFESTATIONS	60
27.	CO-RELATION BETWEEN CO-MORBIDITIES AND CARDIAC MANIFESTATIONS	61

LIST OF FIGURES

Fig. No.	Title	Page No.
a	THE PATHOGENESIS OF MYOCARDIAL INVOLVEMENT IN DENGUE FEVER.	3
1.	AGE DISTRIBUTION	33
2.	GENDER DISTRIBUTION	34
3.	TLC DISTRIBUTION	35
4.	DLC NEUTROPHILS DISTRIBUTION	36
5.	DLC LYMPHOCYTE DISTRIBUTION	37
6.	PLATELET COUNT DISTRIBUTION	38
7.	DISTRIBUTION OF CREATININE CLEARANCE (CRCL)	39
8.	SGOT DISTRIBUTION ACCORDING TO GENDER	40
9.	SGPT DISTRIBUTION ACCORDING TO GENDER	41
10.	RATIO OF SGOT/SGPT	42
11.	CHEST X-RAY (CXR) FINDINGS	43
12.	HEART RATE DURING HOSPITAL STAY	44
13.	ECTOPICS DISTRIBUTION	45
14.	BRADYCARDIA DISTRIBUTION	46
15.	TACHYCARDIA DISTRIBUTION	47
16.	LEFT VENTRICULAR EJECTION FRACTION (LVEF) DISTRIBUTION	48
17.	CREATINE KINASE-MB (CKMB) DISTRIBUTION	49
18.	TROPONIN-T (TRT) DISTRIBUTION	50
19.	D-DIMER DISTRIBUTION	51
20.	MYOGLOBIN DISTRIBUTION	52
21.	LACTATE DEHYDROGENASE (LDH) DISTRIBUTION	53
22.	CARDIAC MANIFESTATION DISTRIBUTION	55
23.	OUTCOME OF CARDIAC MANIFESTATIONS	57

INTRODUCTION

Dengue is caused by an arbovirus belonging to the genus flavivirus, which is transmitted by the bite of female Aedes aegypti and Aedes albopictus mosquitos, which bite during day time. The disease is a major public health problem in the tropical and subtropical belt and according to WHO, dengue is the most important mosquito borne viral disease of the world.[1] The geographical areas affected by dengue have expanded in recent years to include Asia, Africa and Americas. In endemic countries of Asia, the burden of disease is approximately 1300 disability-adjusted life years per million population, which is similar to the disease burden of other childhood and tropical diseases, including tuberculosis, in these regions.[2]

The disease is especially prevalent in India, where insanitary conditions and water logging serve as fertile ground for the mosquito to breed. The disease is rampant in most states of the union and it showed a rising trend from 2015 to 2017. In 2018 there was a marginal decrease in the number of cases and associated deaths. The state of Punjab bears the brunt of the disease with 14980 cases reported in 2018.[3]

The peak season of the disease is from July to November which corresponds to the breeding season of the vector. The dengue arbovirus has four strains labelled as DENV1, DENV2, DENV3 and DENV4 and all four strains are circulating in India. 65% genome is shared between these serotypes and all four serotypes cause identical clinical manifestations in humans. Antibodies to these four strains cross react with each other however, they do not confer immunity against other strains. An effective vaccine against dengue, therefore needs to be polyvalent. As the dengue infection confers sero- specific lifelong immunity, a second attack of dengue can only occur with a different serotype. This second attack is however, potentially more dangerous and associated with a higher rate of complications.[4]

Pathophysiology

The dengue virus replicates in mononuclear cells including immature skin dendritic cells, tissue macrophages, peripheral blood monocytes and hepatocytes. Infected dendritic cells mature and migrate to local or regional lymph nodes where they present viral antigens to T cells and initiate cellular and humoral immune responses. There is replication of the virus in hepatocytes, monocytes and macrophages. The antibodies which are raised against the primary infecting dengue virus serotype may cross react with the subsequent infecting serotype and through opsonization may enhance the ability of second serotype to infect the host. This process is called antibody-dependent enhancement (ADE) of infection. Both macrophages and monocytes participate in antibody-dependent enhancement.[5] ADE occurs when mononuclear phagocytes are infected through their Fc receptors by immune complexes formed by the dengue virus and non-neutralizing antibodies. These non-neutralizing antibodies are a result of previous infection with a different serotype. Infection of mononuclear cells results in the release of inflammatory cytokines and chemokines that produce the manifestations of disease. Therefore, the manifestations of disease depend upon the immunological status of the individual.

The exact mechanism of cardiac injury in dengue is not known, however it is postulated that there is direct invasion of the myocardial cells by the virus which causes inflammatory damage to the myocytes. Dengue virus upon its entry in the body is taken up by the macrophages which causes activation of the T cells. These activated T cells cause release of various inflammatory cytokines, interleukins, tumor necrosis factorand activation of the complement pathway (C3a, C5a) and histamine. This leads to the inflammation and necrosis of the endothelial cells leading to their dysfunction and plasma leakage. Leakage of the plasma in the interstitial space cause myocardial interstitial edema leading to impairment of myocardial function. Decreased fluid in the intravascular compartment secondary to plasma leakage leads to alteration in the coronary circulation. The various

inflammatory markers which are released by activated T cells cause suppression of myocardial contractility and disturbances in the conduction pathway. Various inflammatory markers released cause direct suppression of the cardiac contractility and alteration in the electrical conduction of heart leading to various conduction blocks and ventricular arrhythmia. The release of these inflammatory markers is more in severe form of the disease that correlates with the higher incidence of cardiac manifestations in patients with severe form of the disease. Lactic acidosis which occurs in response to altered hemodynamics also contributes to depression of cardiac contractility.

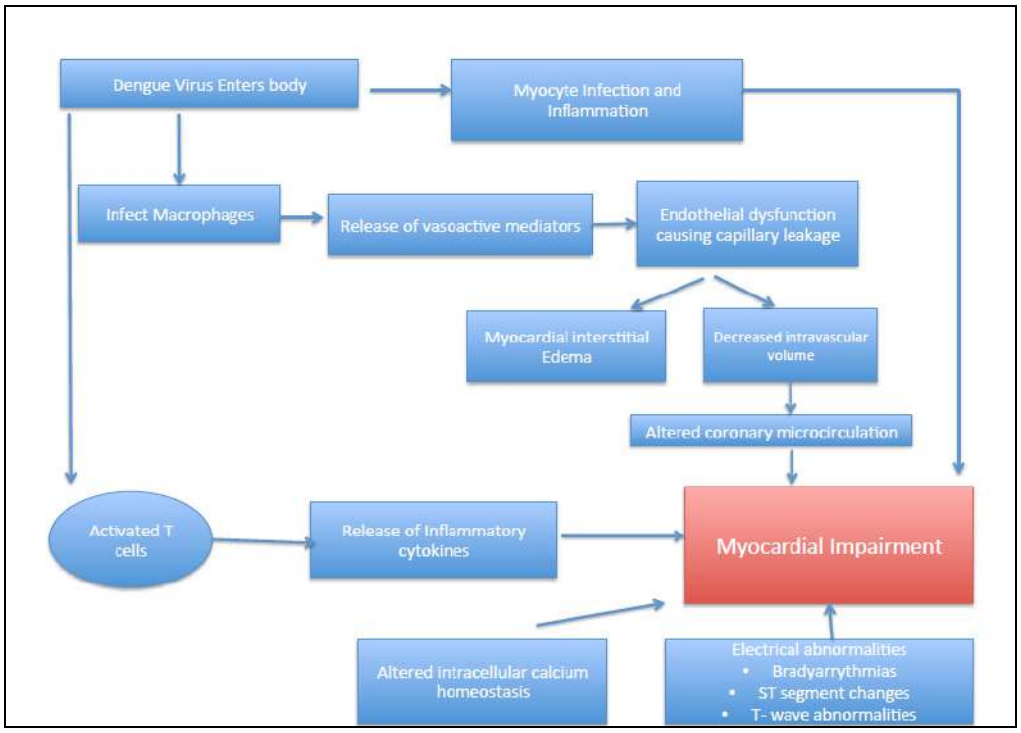

Figure a. The pathogenesis of myocardial involvement in Dengue fever.

CLINICAL MANIFESTATIONS

Majority of dengue cases are subclinical. Symptomatic disease has an incubation period of four to seven days and manifests as high-grade fever, malaise, retro-orbital pain and myalgia. The disease has three clinical phases. The febrile phase, critical phase and the phase of recovery. In the febrile phase there is usually high-grade fever associated with nausea, vomiting and loss of appetite. Fever is usually associated with leucopenia and relative bradycardia. The critical phase begins three days after the onset of fever and lasts for seven days. During this phase there is leakage of fluid in the extracellular spaces with resultant rise in hematocrit and hypotension. The platelets fall during this phase and the condition can progress to shock, coagulopathy and multi organ failure resulting in death.

Dengue fever presents as varied clinical spectrum of dengue fever (DF), dengue haemorrhagic fever (DHF), dengue shock syndrome (DSS), and expanded dengue syndrome (EDS). Most of dengue associated deaths are ascribed to DHF, DSS or EDS. Myocardial involvement occurs in expanded dengue syndrome.

The warning signs of dengue are: -

- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleeding
- Lethargy or restlessness
- Liver enlargement by >2cm
- Increase in haematocrit concurrent with rapid decrease in platelet count

The criteria for severe dengue are: -

- Severe plasma leakage leading to shock (Dengue shock syndrome) or fluid accumulation with respiratory distress
- Severe bleeding
- Severe organ involvement of: -

1) Liver: Increased level of serum aminotransferases of 1000 U/L or more.
2) CNS: impaired consciousness
3) Involvement of heart or other organs.

Cardiac manifestations in dengue fever are common especially in severe form of the disease. According to Moolani et al a considerable component of the burden of cardiovascular diseases is caused by dengue and other neglected tropical diseases.[6]

The various cardiac manifestations include

- Sinus tachycardia
- Sinus bradycardia
- Nonspecific ST-T changes
- SA node dysfunction
- AV dissociation with variable degrees of heart block
- Tachyarrhythmias like atrial fibrillation and ventricular tachycardia
- Pericardial effusion
- Wall hypokinesia
- Elevation of cardiac enzymes

The spectrum of cardiac manifestations in dengue can range from asymptomatic disease to severe myocarditis culminating in death. At present it is not well established if any of the dengue serotypes has a greater propensity to cause cardiac manifestations. However, the incidence of cardiac manifestations is more in the second attack of dengue. Further studies are needed to establish the relationship between serotypes, antigenicity, and cardiac involvement.[7]

The incidence of cardiac manifestations is higher in severe form of the disease.[8] Sometimes however, it may be difficult to attribute abnormalities like arrhythmia to cardiac involvement as these can be seen due to electrolyte imbalance also. However, a detailed clinical examination, ECG, cardiac biomarkers and echocardiography can lead to an accurate diagnosis. The clinical features which suggest cardiac involvement are chest pain, palpitations, hypotension, irregular pulse, rhythm abnormalities, pleurisy and shock. A better understanding of

cardiac complications will potentially improve the treatment of dengue illness by avoiding otherwise preventable morbidity and mortality in the affected patients.

Diagnosis

The diagnosis of cardiac involvement in dengue fever rests upon clinical examination, ECG, cardiac biomarkers and echocardiography.

Kularatne et al used ECG abnormalities to diagnose cardiac manifestations of dengue, although ECG cannot be a stand-alone modality to diagnose these. ECG abnormalities that have been reported with dengue include rate and rhythm abnormalities, heart block, wave form abnormalities, and voltage abnormalities. Reported rhythm abnormalities include relative bradycardia, sinoatrial block, disorders of atrioventricular conduction, second-degree and complete heart block, and monomorphic premature ventricular contractions on a background of heart block, atrial flutter, transient and persistent atrial fibrillation, self-limiting tachy-brady arrhythmia, sinoatrial block, and uniform ventricular ectopics progressing to ventricular bigeminy. There can be ST segment elevation resembling myocardial infarction.[9]

Various biomarkers are used for the detection of cardiac involvement in dengue. These include myoglobin, creatine kinase-muscle brain-type, N-terminal pro-brain natriuretic peptide, heart-type fatty acid-binding protein and troponin-T. Although biomarkers may diagnose the cardiac involvement early, but, they do not show a good correlation with cardiac function.

Echocardiography is an important tool to diagnose cardiac function. Indications for echo include ECG changes and clinical signs of heart failure or shock. It can evaluate chamber dilatation, systolic and diastolic dysfunction, wall motion abnormalities as seen in myocarditis and also assess left ventricular ejection fraction (LVEF). Pericardial effusion can also be easily picked up on echocardiography. Myocardial performance index or Tei index can also be calculated [10]. Echo provides a good insight into the hemodynamics of dengue patients by measuring the preload, LVEF and myocardial performance index. In

patients with resistant shock the presence of myocarditis should be suspected and cardiac magnetic resonance imaging (MRI) may also be helpful in such cases.

Management

As the cardiac insult is usually mild and transient, in most cases no specific treatment may be required for its management. Severe cases require symptomatic treatment and correction of electrolyte imbalance in order to maintain hemodynamic stability. Supportive care includes intravenous fluids to prevent hemodynamic alterations, although fluid overload should be avoided at all costs in order to prevent pulmonary edema.

Determining whether shock is due to hemodynamic alteration or myocarditis remains a clinical dilemma. However, echocardiography can diagnose myocarditis and help resolve this dilemma. Atrial fibrillation is the most commonly clinically encountered arrhythmia, although it is rare. This may require treatment with anti-arrhythmic drugs.

Inotropic agents are used to treat depressed cardiac contractility and cardiac pacing may be required to treat persistent heart blocks [11].

Early recognition of myocardial involvement in dengue illness,prompt restoration of hemodynamic instability while avoiding fluid overload, and sparing unnecessary invasive management areimportant in treating dengue-affected patients with severe myocarditis.[12]

The government of India has issued national guidelines for the management of dengue [13]. According to this, dengue fever has been divided into mild, moderate and severe types for proper management. Mild dengue refers to fever without any complications and without capillary leakage. This can be managed at home or OPD level. Moderate dengue refers to fever with high-risk co-morbid conditions or fever with warning signs or mild bleeding. Such patients need close monitoring and hospitalisation. Severe dengue refers to dengue with severe haemorrhage with or without shock or expanded dengue

syndrome or metabolic acidosis. Such patients need tertiary level care.[13]

AIMS & OBJECTIVES

1. To study the spectrum of cardiac manifestations in dengue fever.
2. To ascertain the impact of cardiac abnormalities on patient outcome.

REVIEW OF LITERATURE

In 1998 Wali et al studied cardiac involvement in cases of dengue hemorrhagic fever. This study was undertaken at AIIMS New Delhi. They investigated 17 patients of dengue hemorrhagic fever and dengue shock syndrome for cardiac involvement with radionuclide ventriculography, echocardiography and ECG. They detected cardiac involvement in the form of reduced ejection fraction and global hypokinesia. However, all these changes were reversible and Tc 99m pyrophosphate scans revealed no evidence of myocardial necrosis in the study group. The authors concluded that reversible cardiac involvement may occur in dengue hemorrhagic fever or dengue shock syndrome and this may account for unexplained hypotension in these patients.[14]

In 1998 a study was published by Kabra et al regarding myocardial dysfunction in children with dengue hemorrhagic fever. This study was done at All India Institute of Medical Sciences during the 1996 dengue epidemic. WHO treatment guidelines favored fluid replacement therapy to treat hypotension.This study was undertaken to find if other factors like myocardial dysfunction also play a role in the etiopathogenesis of hypotension. They did echocardiography on 54 children presenting with hypotension and found reduced ejection fraction in 9 (16.7%) children. Myocardial involvement was found to be independent of the severity of disease. They advocated that myocardial function must be assessed in patients who have persistent hypotension in spite of adequate hydration.[15]

The dengue epidemic of 1996 was also the subject of study by Agarwal et al who published a paper in 1999 and described the clinical findings of dengue and dengue hemorrhagic fever in the city of Lucknow. They found that age group of 11 to 30 years was the most affected with the male female ratio of 1.9:1. The most common symptom was frontal headache followed by myalgia, skin rash and vomiting. Out of 206 patients studied cardiac involvement was seen in only 1 patient. The

most common site of bleeding observed in DHF was from the gums. They observed a mortality of 47% in cases who had profound shock.[16]

In 2003 Khongphatthanayothin etal studied left ventricular performance and hemodynamic abnormalities in different stages of dengue hemorrhagic fever. The study was carried out in a tertiary medical school hospital. 24 patients were included in the study who had serologically confirmed dengue and dengue hemorrhagic fever (DHF). They found that rate-corrected velocity of circumferential fiber shortening adjusted for end-systolic meridional wall stress (VCFC/ESS) and ejection fraction were considerably less in the toxic stage. They concluded that cardiac output decreases by a complex mechanism in DHF. Decreased preload coupled with reduced ejection fraction and subnormal heart rate are primarily responsible for it.[17]

Promphan et al in 2004 reported a case of a 13-year-old Thai boy who was suffering from bradycardia. The boy had history of high-grade fever and he developed hypotension, ascites and bilateral pleural effusion after 4 days of fever. The liver was enlarged and tender. The boy recovered from shock on day 5 after meticulous fluid management and did not suffer from any bleeding diathesis. On the 6th day of illness, he developed hypotension and bradycardia. His slowest heart rate was 50 beats/minute in a junctional escape rhythm seen on 12-lead electrocardiography (ECG). At the beginning, adrenaline was infusedat a rate of 0.2 microgm/kg/minute in the ICU. His heart rate increased to 80 beats/minute in a junctional rhythm and became hemodynamically stable. Then he developed frequent episodes of premature ventricular contractions (PVCs), therefore lidocaine 1 mg/kg was intravenous administered followed by a continuous infusion at 16 μg/kg/minute. He was referred to tertiary care hospital on day 7 of illness. He had cold clammy skin, fair tissue perfusion, slightly puffy eyelids, and mild hepatomegaly. Cardiac auscultation revealed an S3 gallop without a significant murmur. An electrocardiogram demonstrated sinus tachycardia, nonspecific ST-T change, and no PVCs. A CBC demonstrated a hematocrit of 32%, white blood cells

8,100/mm^3 (neutrophils 71%, lymphocytes 18%), and platelets 39,000/mm^3. Echocardiography showed impaired left ventricular systolic function with an ejection fraction of 40%. He was transferred to the pediatric intensive care unit for close monitoring. Neither arrhythmias nor hypotension occurred, and the adrenaline and lidocaine were successfully discontinued on the next day. A follow-up echocardiographic study on day 14 of the illness showed LV ejection fraction of 68%. Prednisolone 2 mg/kg/day was started on day 16 of the illness. He was discharged after 7 days of admission with digoxin, furosemide, and prednisolone 2 mg/kg/day. His ECG at the time of follow-up showed normal sinus rhythm. The prednisolone was eventually tapered off 10 days after his first dose. The authors concluded that their patient developed dysfunction of the sinus node and impairment of ventricular systolic performance a day after recovery from the dengue shock stage, which is an unusual pattern of cardiac involvement in dengue patients. The impairment of left ventricular systolic function was ascribed to myocardial damage or myocarditis.[18]

Singhi et al in 2007 described the epidemiology, clinical features and treatment of dengue fever and dengue shock syndrome. They searched the literature on Pubmed, World Health Organization (WHO) and PAHO websites using the terms dengue and dengue shock syndrome. They complemented this information with personal practice to write about dengue. According to the authors, dengue is the most important arthropod-borne viral disease of humans. Its presentation is protean and varies from an undifferentiated viral syndrome to hemorrhagic fever and severe shock. Dengue fever is a self-limiting, nonspecific illness characterized by fever, headache, myalgia, and constitutional symptoms. Its severe forms (hemorrhagic fever and shock syndrome) may lead to multisystem involvement and death. Early diagnosis, close monitoring for deterioration and response to treatment are necessary in all cases. WHO has provided a step wise approach to management that is useful for milder forms and early shock. In the more severe forms aggressive fluid resuscitation and

support for failing organs is necessary for the critically ill patient. Research addressing pathophysiological differences between dengue shock and septic shock, choice of fluids, inotropes and techniques of organ support are likely to yield benefits for the critically ill. The authors felt that there was no specific therapy for dengue infections. Good supportive care was lifesaving, but ultimately initiatives aimed at vector control and prevention of mosquito bites would provide the maximum benefits.[19]

Satarasinghe et al in 2007 undertook a study regarding the recent surge of dengue viral cases in Sri Lanka especially dengue hemorrhagic fever. They studied myocardial involvement in clinically and serologically confirmed cases of dengue infection. They studied 217 cases of dengue fever in the age group 12 to 65 years. 185 patients underwent echo examination and myocarditis was detected in 24% cases. All cases of myocarditis had bradycardia and one patient had Wenckebach phenomenon. There was no mortality in their study group. On follow up echocardiographic study there was complete resolution of myocarditis. The authors postulated that myocardial involvement is transient and self- limiting in dengue fever.[20]

Lateef et al in 2007 observed that bradycardia was a notable clinical feature of dengue in Singapore outbreak of 2005. They claimed that this sign was not previously associated with dengue and awareness of this clinical finding could help in the early recognition of dengue and potentially reduce complications and death associated with dengue virus infection.They performed a case-control study comparing febrile dengue patients to patients with other infectious diseases. Their study demonstrated a consistently lower heart rate at all peak temperatures recorded in dengue patients. According to authors relative bradycardia occurs in many infectious diseases, including typhoid fever, Legionnaire's disease, psittacosis, typhus, leptospirosis, malaria, and babesiosis. Fever production in response to exogenous pyrogens is mediated mostly by cytokine prostaglandin pathways, and neural input is important in the early phases of fever. Concentrations of cytokines,

including tumor necrosis factor, interferon-γ, interleukin-8 (IL8), IL-10, and IL-12, are substantially increased during dengue infection. Their levels likely correlate with specific clinical manifestations and illness severity. However, the relationship of cytokines to relative bradycardia in dengue could not be established by the authors.[21]

In 2007, Kularatne et al described a high incidence of cardiac complications in an outbreak of dengue fever at General Hospital, Peradeniya, Sri Lanka, in 2005. They studied 120 serologically confirmed dengue cases and 75 of these patients (62.5%) had changes on electrocardiogram in the form of T inversion, ST segment depression and bundle branch blocks. These were assigned to the 'cardiac group'. The remaining 45 patients did not have any changes on the electrocardiogram and were assigned to 'non-cardiac group'. It was observed that the cardiac group patients had greater preponderance of symptoms like fatigue, dyspnea, low peripheral oxygen saturation, chest pain and flushing of skin. In the cardiac group, 17 (23%) patients had hypotension and 58 (77%) developed tachycardia and bradycardia indicating significant cardiac dysfunction. No correlation between pulse rate and body temperature was observed in the study.[22]

In 2007, Wang et all in a retrospective study, studied 363 dengue hemorrhagic fever cases in southern Taiwan and reviewed a total of 468 chest X- rays. More than 50% showed abnormalities after the 3rd day and pleural effusion and infiltration were the only major findings. Infiltration was detected in 23.3% cases and pleural effusion was observed in 25% cases. Infiltration was bilateral in 45% cases. Pleural effusion was observed on the right side in majority of cases.[23].

Mahmod et al in 2009 described a case of atrial fibrillation in a 20-year-old patient of DHF.The diagnosis of DHF was established by characteristic clinical and laboratory features of fever, hematological abnormalities (thrombocytopenia, leukocytopenia, and hemoconcentration), evidence of plasma leakage (postural hypotension), and positive serological tests. The patient developed

hemodynamically stable atrial fibrillation (AF) which persisted even after the fever had subsided. Therefore, on follow up after one week, the patient was started with oral amiodarone and warfarin and he reverted to sinus rhythm. According to the authors, although self-limiting cardiac complications are common in dengue, AF is rare. The AF was attributed to dengue infection because the patient did not have any underlying cardiac abnormality like valvular heart disease or cardiomyopathy. This case was different as the AF was not self-limiting, but required anti-arrhythmic treatment. The authors advocate antiarrhythmic treatment like cardioversion or oral pharmacological treatment for arrythmias which are not self-limiting. [24]

Lee et al in 2010 reported a case of 64-year-old woman who developed myocarditis in DHF. The condition mimicked myocardial infarction as there was elevation of cardiac specific troponins and depression of ST-segment in precordial leads. In a review of literature, the authors found that cardiac manifestations in dengue varied widely from self-limiting tachy or brady arrhythmias to fatal myocarditis. According to the authors the incidence of cardiac manifestations in dengue is under-reported as most cases are mild and clinically insignificant. The authors feel that early diagnosis of cardiac complications with initiation of proper treatment is important for favorable patient outcome. Fluid overload and unnecessary invasive procedures must be avoided in dengue patients.[12]

Kaushik et al in 2010 described the case of a 10-year-old girl who presented with complaints of fever, abdominal pain and vomiting of one week's duration. There was a petechial rash over the patient's back, trunk and upper extremities. The Hess tourniquet test for capillary fragility was positive. Platelet count was low (38000/cumm). Dengue serology was reactive for immunoglobulin G (IgG) and immunoglobulin M (IgM). The patient was managed supportively according to the World Health Organization (WHO) recommendations for management of dengue hemorrhagic fever. During the course of illness, the patient developed bradycardia (pulse rate 50/min, regular), with normal blood

pressure and normal heart sounds. Electrocardiography (ECG) revealed a rate of 44/min and varying PP intervals (an absence of normally expected P waves), which was suggestive of SA block, with no relation (dissociation) between P waves (atrial activity) and QRS complex (ventricular activity) indicative of AV dissociation. Echocardiography revealed normal cardiac valves, left ventricular systolic and diastolic functions, and cardiac chamber dimension. There was no evidence of pericardial effusion or vegetations. In the presence of rhythm disturbances, the patient was further investigated for underlying myocarditis. The creatinine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels were 172 U/dL (normal < 130 U/dL) and 776 U/dL (normal < 330 U/dL), respectively. The serum potassium and calcium levels were found to be normal. The patient was asymptomatic, and her vital signs were closely monitored for the development of any complications. She was discharged after eight days, with a heart rate of 52/min and a normal BP, and the ECG showed a persisting complete AV block. The patient was kept on close follow-up for one week, during which her bradycardia and complete AV block persisted. A follow-up after five months showed a normal heart rate (82/min). ECG reverted to a normal sinus rhythm, and the echocardiography was also normal. The presence of bradycardia and elevated cardiac enzyme levels (LDH and CPK) was consistent with a diagnosis of myocarditis, although there were no signs of cardiogenic shock or congestive heart failure. The co -existence of SA block with AV dissociation on ECG could possibly explain the rhythm abnormalities associated with myocarditis in their patient. Dengue virus -induced myocarditis could apparently explain the SA block and AV dissociation observed in this case. Approximately 40% patients recover from the rhythm abnormality in myocarditis. The authors postulated that patients with fewer cardiac symptoms have a better prognosis and that complete resolution can be expected in such cases. Acute high-grade AV blocks occasionally respond to atropine and isoproterenol. These drugs were not used in this patient due to the dangerous slow escape

rhythms associated with them. In view of the inadequate escape rhythms and persistent bradycardia, temporary cardiac pacing was advised for this patient, which the patient declined due to cost constraints. However, spontaneous resolution was observed after five months. In the authors' experience, the rhythm abnormalities in dengue fever tend to be benign and self -limited, and resolve in the majority of patients at discharge or on follow-up. [25]

Salgado et al in 2010 undertook a prospective study of 102 children with DHF from Colombia, South America. Clinical diagnosis of myocarditis was made by a paediatric cardiologist and confirmed by ECG and echocardiography. Immunohistochemistry for detection of dengue virus and inflammatory markers was performed on autopsied heart tissue. In vitro studies of human striated skeletal fibers (myotubes) infected with dengue virus were used as a model for myocyte infection. Measurements of intracellular Ca^{2+} concentration as well as immunodetection of dengue virus and inflammation markers in infected myotubes were performed. Eleven children with dengue haemorrhagic fever had presented with symptoms of myocarditis. Widespread viral infection of the heart, myocardial endothelium, and cardiomyocytes, accompanied by inflammation was observed in 1 fatal case. Immunofluorescence confocal microscopy showed that myotubes were infected by dengue virus and had increased expression of the inflammatory genes and protein IP-10. The infected myotubes also had increased intracellular Ca^{2+} concentration. The authors concluded by expressing that vigorous infection of heart tissues in vivo and striated skeletal cells in vitro along with derangements of Ca^{2+} storage in the infected cells directly contributed to the presentation of myocarditis in pediatric patients.[26]

Gupta and Gadpayle did a retrospective study in 2010 to study cardiac involvement in dengue hemorrhagic fever. Their study comprised of 28 patients of dengue haemorrhagic fever (DHF) admitted at PGIMER, Dr R. M. L. Hospital, New Delhi, between July 2008 to October 2008. Dengue was confirmed as per WHO criteria and all patients were IgM

positive on Elisa. All patients were investigated with CBC (including PCV and platelet counts), ECG, ECHO, cardiac enzymes (CPK, CPK-MB, TROPONIN-T, SGOT, LDH), USG abdomen, and X-ray chest. None of their patients had clinical features of overt myocarditis. 4 patients had sinus bradycardia and 6 cases had sinus tachycardia. There were no other ECG abnormalities. Out of 28 patients of DHF, 20 patients (71%) had significantly raised cardiac enzymes CPK-MB, LDH and SGOT. 12 patients tested positive for serum Troponin-T. Four patients (14%) had grade 1 diastolic dysfunction in Echo, and one patient (3.5%) had mild pericardial effusion. The authors concluded that a significant number (71%) of patients of dengue developed asymptomatic involvement of heart as evidenced by raised cardiac enzymes (CPK-MB, Troponin-T, LDH and SGOT). Myocardial involvement was subclinical as Echo was normal in 23 patients (82%). The authors postulated that raised cardiac enzymes in asymptomatic patients were possibly due to subclinical myocarditis.[27]

La-Orkhun V et al in 2011 studied cardiac rhythm abnormalities and heart rate variability during convalescent stage of dengue virus infection in children. During the convalescent stage, cardiac rhythm abnormalities were found in ten patients (29%), including sinus pause (1), first-degree (2) and Mobitz type I second-degree AV block (Wenckebach) (3) and atrial (4) and ventricular ectopic beats (5). There was no relationship between the clinical severity of dengue virus infection (DF, DHF without shock and DSS) and the incidence of cardiac arrhythmia. The authors concluded that various benign bradyarrhythmias and ectopic beats can be detected in patients with dengue virus infection during the convalescent stage.[28]

Weerakoon et al published a paper in 2011 on the outbreak of dengue in Sri Lanka in 2009, which caused a high fatality. They conducted 5 autopsies of clinically suspected myocarditis cases at the General Hospital, Peradeniya to describe the histopathology of the heart and other organs. The diagnosis of dengue was confirmed with specific IgM and IgG ELISA, HAI and RT-PCR techniques. The histology was done

in tissue sections stained with hematoxylin and eosin. Of the 319 cases of dengue fever, 166(52%) had severe infection. Of them, 149 patients (90%) had secondary dengue infection and in 5 patients, DEN-1 was identified as the causative serotype. The clinical diagnosis of myocarditis was considered in 45(27%) patients. The autopsies were done in 5 patients who had severe hypotension and succumbed to shock (3 females and 2 males) aged 13- 31 years. All had pleural effusions, ascites, bleeding patches in tissue planes and histological evidence of myocarditis. The main histological findings of the heart were interstitial oedema with inflammatory cell infiltration and necrosis of myocardial fibers. One patient had pericarditis. The concurrent pulmonary abnormalities were septal congestion, pulmonary hemorrhage and diffuse alveolar damage; one case showed massive necrosis of liver. The authors proved conclusively that dengue can cause myocarditis which can be potentially fatal.[29]

Azin et al in 2012 undertook a study to correlate laboratory tests during the evolution of dengue fever. Their study was descriptive and retrospective conducted in a referral centre for infectious diseases. The tests analyzed were blood count, platelet count, and serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) concentrations. Thrombocytopenia and elevated transaminases were observed in patients with classic dengue fever. Most laboratory abnormalities started on the 3rd day but were more evident on the 5th day with restoration of values by the 11th day; this was more prominent in under 15-year-olds and with the more severe clinical forms. The authors observed leucocytosis in the first few days of the disease followed by leucopenia. Similarly, there was a decrease in lymphocytes in the early days of disease followed by lymphocytosis.[30]

Yacoub et al in 2012 studied seventy-nine patients aged 8-46 years with different dengue severity grades using echocardiography including tissue Doppler imaging. The patients were divided into severity grades of dengue, dengue with warning signs and severe dengue. Changes in cardiac functional parameters and haemodynamic indices were

monitored over the hospital stay. Patients with severe dengue had worse cardiac function compared to dengue, in the form of left ventricular (LV) systolic dysfunction with increased Left myocardial performance index. Septal myocardial systolic velocities (S′) were reduced as well as right ventricular (RV) systolic and diastolic (E′) velocities. The authors concluded that patients with severe dengue had evidence of systolic and diastolic cardiac impairment, with predominant involvement of septal and right ventricular wall.[31]

Miranda et al in 2013 performed a study on 81 confirmed cases of dengue. They estimated the value of cardiac biomarkers like troponin I and N terminal fragment of B-type natriuretic peptide. Patients with abnormal biomarkers underwent echocardiography. Patients who had abnormal echo findings further underwent cardiac magnetic resonance imaging. The authors found that cardiac manifestations had no correlation with the clinical dengue classification or the severity of dengue infection. It was also irrespective of duration of symptoms or prevalence of secondary infection. Two of their patients died of cardiogenic shock which autopsy proved, to be due to myocarditis. 10 patients were subjected to echocardiography. Out of these 10 patients 1 patient had decreased left ventricular ejection fraction (LVEF), left ventricular segmental abnormalities with preserved LVEF were observed in 2 patients and pericardial effusion with tamponade was observed in 1 patient. These 4 patients showing abnormalities on echocardiography were made to undergo cardiac MRI examination and the findings were confirmed on same. The authors concluded that cardiac manifestations in dengue can range from asymptomatic elevation of cardiac biomarkers to life threatening myocarditis or pericarditis. Failure to diagnose cardiac complications can result in significant patient morbidity and mortality. [32]

Tarique et al in 2013 studied the ECG changes in patients with dengue fever and dengue hemorrhagic fever in the department of Medicine, Mayo Hospital Lahore. 116 patients with dengue infection were enrolled in the study. Their clinical presentation and examination were

duly noted. Each patient had baseline and then regular monitoring of blood counts, metabolic profile and fluid status. Patients with Dengue Hemorrhagic fever underwent radiological examination in form of chest radiograph and ultrasound abdomen. ECG was carried out in all patients. Out of 116 patients, 61(52.6%) suffered from Dengue Fever and 55(47.4%) had Dengue Hemorrhagic Fever. Overall, 78 patients had normal ECG. Abnormal ECG findings like tachycardia, bradycardia, supraventricular tachycardia, left bundle branch block, ST depression, poor progression of R wave were noted. The authors concluded that ECG changes can occur in dengue infection with or without cardiac symptoms and common ECG changes were ST depression and bradycardia.[33]

Sam et al in 2013 described in their study, the outbreak of dengue fever in Malaysia in 2006 to 2007. This outbreak caused a large number of fatalities. The authors undertook a retrospective study to examine dengue death cases in their hospital from June 2006 to October 2007 with a view to determine if there had been changes in the presentation of severe to fatal dengue. Nine of the ten fatal cases involved adult females with a median age of 32 years. All had secondary dengue infection. The mean duration of illness prior to hospitalization was 4.7 days and deaths occurred at an average of 2.4 days postadmission. Gastrointestinal pain, vomiting, diarrhea, intravascular leakages and bleeding occurred in the majority of cases. DSS complicated with severe bleeding, multi-organ failure and coagulopathy were the primary causes of deaths. Seven patients presented with thrombocytopenia and hypoalbuminemia, five of which had hemoconcentration and raised serum transaminases indicative of liver damage. Prominent unusual presentations included acute renal failure, acute respiratory distress syndrome, myocarditis with pericarditis, and hemorrhages over the brain and heart. The authors concluded that uncommon presentations of dengue were a reflection of the changing demographics where adults rather than children, were more likely to contract dengue in dengue endemic regions.[34]

Sengupta et al in 2013 studied 40 subjects with two-dimensional (2D) speckle tracking echocardiography to analyze left ventricular (LV) myocardial performance. Of these 40 subjects, 20 patients had dengue hemorrhagic fever (DHF) with thrombocytopenia and 20 were healthy controls. Images of the left ventricle in the four- three- and two-chamber views and parasternal short-axis views at the basal, mid and apical levels were obtained. Of the 20 patients, imaging was performed again in 19 at discharge after a hospital stay of approximately 8 days. Longitudinal, circumferential and radial strains were quantified and compared in an 18- segment model using a novel speckle tracking system. It was observed that left ventricular global ejection fraction was reduced in patients with DHF at presentation as compared with controls. Peak longitudinal strain in patients with DHF was significantly attenuated in the subendocardial region compared with normal controls. A significant increase in circumferential strain for patients with DHF was evident only in the subepicardial region. Patients with DHF showed significantly higher radial strain than controls. On multivariate analysis, subendocardial longitudinal strain independently predicted the duration of hospital stay in patients with DHF. It was concluded by the authors that assessment of speckle tracking echocardiography- derived LV mechanics helped in understanding myocardial mechanics in patients with DHF and thrombocytopenia. Identification of reduced LV longitudinal strain helped in understanding the mechanism of reduced LV myocardial performance seen in patients with DHF.[35]

In 2013, Marques et al reported two cases of dengue myocarditis involving young adult males. The first case presented with typical signs of myocardial disease i.e., chest pain and diaphoresis with myocardial depression in the electrocardiograph. The second case deteriorated rapidly and demised within the first day of admission. Histology of the heart muscles showed multiple small foci of myocyte necrosis surrounded by lymphocytes, in keeping with viral myocarditis. Both cases fulfilled the World Health Organization (WHO) diagnosis of probable dengue: the first case had positive dengue serology, both IgM

and IgG at day six of illness, and the second case was polymerase chain reaction (PCR) positive for dengue and identified as serotype 2. Although both cases did not fulfill the criteria of DHF, still both had severe morbidity and cardiac manifestations. The authors felt that severe cardiac impairment is not uncommon in dengue fever and can be life threatening.[36]

Mallhi et al performed a retrospective analysis of dengue patients in a tertiary care hospital. The study examined the patients during a period of six years, from 2008 to 2013 and it was published in 2015. They categorized patients into two groups of dengue fever (DF) and dengue hemorrhagic fever (DHF). These groups were compared to determine the clinical and laboratory features and also to determine the risk factors of DHF. They studied 667 patients and observed DHF in 79 patients (11.8%) They observed that skin rash, dehydration, dyspnea, pleural effusion and thickened gall bladder wall were observed more commonly in DHF. Statistical analysis demonstrated that age more than 40 years, secondary infection, diabetes mellitus, lethargy, thick gall bladder wall and delayed hospitalization were independent predictors of DHF. They observed an overall mortality of 1.2% in their study. The authors concluded that predictors of DHF allowed the clinician to identify patients at high risk, thus giving adequate time to the clinician to plan for appropriate treatment.[37]

In 2015, Shivanthan et al performed a systematic review of MEDLINE, EMBASE, Scopus, SciVerse, Google Scholar, and LILACS for papers on cardiac involvement in dengue. They found that cardiac involvement in dengue is common though often transient and self-limiting. However, it has the potential of causing significant morbidity and mortality. A variety of mechanisms were postulated to cause myocardial dysfunction like direct viral invasion, immune mechanisms and electrolyte imbalance etc. Cardiac involvement was assessed by clinical features, cardiac enzymes, electrocardiography and echocardiography. The authors found echocardiography to be an excellent tool for assessing cardiac involvement. In the experience of

the authors, treatment was largely supportive. It was however felt that further studies were required to know more about the pathophysiology and treatment of cardiac manifestations in dengue.[38]

Kirawittaya et al in 2015 studied children less than 15 years of age who were hospitalized for suspected dengue at Queen Sirikit National Institute of Child Health, Bangkok, Thailand in 2010 to 2012. Criteria for suspected dengue included cases with febrile illness without an obvious focal source of infection and with compatible laboratory findings of leucopenia or thrombocytopenia. Patients with chronic hematologic or immunologic conditions were excluded. Patients were treated according to World Health Organization (WHO) guidelines. The authors enrolled 181 dengue cases. Cardiac function was studied in these cases by serial echocardiography. Plasma leakage was detected by serial chest and abdominal ultrasonography. Cardiac troponin-T levels were measured daily. It was found that dengue cases that already developed plasma leakage had lower cardiac index and higher left ventricular myocardial performance index and systemic vascular resistance compared to those without plasma leakage. Early diastolic wall motion of the left ventricle was decreased in dengue cases with plasma leakage compared to those without. Decreased left ventricular wall motility was more common in dengue patients compared to non-dengue cases particularly in cases with plasma leakage. Differences in cardiac function between DF and DHF were most pronounced around the time of plasma leakage. Cardiac dysfunction was transient and did not require treatment. Transient elevated troponin-T levels were more common in DHF cases compared to DF. The authors concluded that transient left ventricular systolic and diastolic dysfunction was common in children hospitalized with dengue and was related to severity of plasma leakage. The functional abnormality spontaneously resolved without specific treatment and cardiac structural changes including myocarditis were uncommon.[39]

Lin et al in 2016 reported the case of a 43-year-old man who suffered from dengue and had ST segment elevation in leads V1 to V5.

Coronary angiography revealed subtotal occlusion of LAD and left circumflex (LCx) arteries for which thrombus aspiration and stenting of both arteries was performed. The authors postulated that the possible cause of vessel occlusion was dengue infection leading to the rupture of multiple unstable plaques. According to authors STEMI is a rare complication of dengue and needs appropriate treatment for favorable patient outcome.[40]

Dhanoa et al in 2016 explored the clinical impact of dengue virus co-infections on clinical manifestations and laboratory tests. This was a retrospective study performed in serologically (PCR) confirmed cases of dengue. They studied a total of 262 patients and forty of them had dengue virus co-infections (15.3%). Secondary infections were observed in 31.3% of patients. Whole genome sequences of random samples representing DENV-1 and DENV-2 showed heterogeneity amongst the Dengue viruses. Statistical multivariate analysis showed that warning signs of dengue were much more common in the group which had co-infection. DENV2 co-infection had higher frequency of severe thrombocytopenia, whereas DENV2 mono-infection presented most commonly with myalgia. Patients with co-infection had also increased incidence of raised creatinine. The authors concluded that dengue co-infections are common in endemic zones and such patients have more severe clinical manifestations.[41]

In 2016, Wei et al studied the dengue epidemic of 2014 in Taiwan which affected more than 15000 people. This epidemic had 136 cases of DHF with 20 fatalities. Median age of DHF was 71 years and most of them had co-morbidities like diabetes mellitus and hypertension. Only half the cases of DHF (59/136) were classified as severe according to 2009 WHO-revised dengue classification. The fatality rate for DHF was 14.7%. Co-morbidities with severe plasma leakage were identified as independent risk factors for fatality. [42]

Virk et al in 2016 reported a case of 31-year-old female who presented to the Emergency Department of Mount Sinai St. Luke's and Mount Sinai Roosevelt Hospitals with low grade fever, headaches,

arthralgia/myalgia, and fatigue one day after returning from a trip to Brazil. On serology, dengue virus specific IgM antibodies were positive and the IgG titer during active infection was 1:1160. The patient developed a third-degree atrioventricular block, with pulse less polymorphic ventricular tachycardia with a run of torsade. A return of spontaneous circulation was achieved after three cycles of CPR including defibrillation, administration of epinephrine and intravenous (IV) magnesium. Patient was resuscitated and a temporary trans-venous pacemaker was placed. She reverted back to normal sinus rhythm after 4 days of syncope and was subsequently discharged from the hospital after complete resolution of symptoms, without the need for a permanent pacemaker. According to the authors, cardiac manifestations and myocardial compromise are more severe in DHF and DSS as compared to dengue fever. The authors also felt that early diagnosis of dengue hemorrhagic fever related cardiac complication was essential and physicians should focuson ruling out other reversible causes of cardiac dysfunction. Meticulous fluid management was considered to be extremely important. Authors also stressed the need to avoid unnecessary invasive procedures like permanent pacing, given the transient nature of cardiac complications in dengue fever.[43]

Yadav and Kumar undertook a study in 2017 to detect ECG changes in patients presenting with dengue infection to evaluate whether there were any clinical cardiac manifestations and to find out the correlation of cardiac manifestations to warning signs of dengue. The study was conducted at MLN Medical College, Allahabad in the Department of Medicine between July 2016 to December 2016. One hundred consecutive patients aged 18 years or more with positive dengue serology were examined. ECG was done for all patients. Thirty-six patients had no warning signs, 64 patients had warning signs, out of which 43 patients had two or more warning signs while 21 patients had single warning sign. Most common warning sign in this study was abdominal pain (50%) and Vomiting (45 %) while hepatomegaly was the least common warning sign. The most common cardiac

abnormalities noted were rhythm abnormalities of which the commonest was sinus bradycardia, found in 60 percent. The authors concluded that there was statistically significant correlation between cardiac manifestations and all the warning signs except lethargy/restlessness and hepatomegaly.[44]

Yadav et al in 2017 studied 104 cases of dengue to assess the magnitude of cardiac involvement in dengue. This study was conducted in the Department of General Medicine, S.M.S. Medical College Jaipur. Out of 104 dengue patients, abnormal ECG findings were observed in 28 patients. The most common ECG finding was sinus bradycardia in 9 patients (32.14%), next was sinus tachycardia in 7 patients (25%) and T-wave inversion in V1- V4 and V2-V5 was observed in 4 and 2 patients respectively. ST depression in V1-V4 and V1-V6 was identified in 3 patients each. 3 patients had generalized T wave inversion and low voltage. ST-T changes in V2-V5 and V3-V6 both were present in 2 patients each, generalized ST elevation and T wave inversion in lead 2, 3 were in 1 patient each. Among these 28 patients, abnormal echo finding was present in 9 patients (32.14%). The most common finding was mild pericardial effusion in 5 patients (17.86%). Among these 104 patients, the mean Value of CK-MB was significantly higher in the cases with cardiac involvement as compared to normal.Out of 104 patients, 9 patients (8.65%) were troponin T positive and 95 (91.35%) were troponin T Negative. Out of 104 patients, 39 patients (37.5%) had elevated CK-MB and 28 patients (29.92%) had abnormal ECG findings. Abnormal 2-D echo and abnormal Troponin T was observed in 9 patients (8.65%) each. The authors postulated that asymptomatic cardiac involvement in dengue is common. Although ECG and cardiac enzymes may be abnormal in such patients, echocardiography reveals normal ejection fraction and such patients have asymptomatic myocarditis.[45]

Kularatne et al in 2018 described dengue as a dreaded arboviral infection with global importance. Presentation of dengue can be with a diverse clinical spectrum ranging from asymptomatic infection or simple

undifferentiated fever to DHF with multiorgan failure. They studied 10 cases of dengue who presented at Teaching Hospital, Peradeniya, Sri Lanka during the dengue epidemic of 2016. This group had seven cases of DHF. The authors observed that bleeding manifestations were seen both in dengue fever and dengue hemorrhagic fever and the cardinal feature that differentiated dengue fever from DHF was not hemorrhage but leaking. Pathological hallmark of dengue hemorrhagic fever was increased capillary permeability with extravasation of fluids during the critical phase of dengue fever. Potential life-threatening complications in their study were bleeding into gut, severe hepatitis, septic shock, myocarditis and intracranial bleeding. These patients were treated with blood transfusions and meticulous fluid management to suit individual needs besides other empirical therapeutic measures. In this study, a young female had evidence of myocarditis and echo revealed left ventricular global hypokinesia. She was managed conservatively with fluids and short course of steroid therapy. Steroids reduced inflammation in her myocardium and improved contractility. Her follow up echocardiography after two weeks showed that her left ventricular function had returned to normal. In the authors experience dengue was an extremely challenging condition to treat. Unusual presentations and complications could be fatal in dengue if not treated timely. The authors acknowledged that bed-side ultrasound examination was a big help in early detection of complications. Theyalso felt that clinicians should be aware of these complications as these were often under-reported and over-looked.[46]

Papalkar et al in 2019 studied the prevalence of cardiac manifestations in dengue period in a tertiary care hospital. This was a cross-sectional study undertaken over a period of two years from September 2016 to October 2018. Serologically confirmed cases of dengue were classified according to the World Health Organization criteria into dengue fever, dengue hemorrhagic fever (DHF), and dengue shock syndrome (DSS). The assessment of cardiac manifestations was done based on electrocardiogram (ECG), echocardiogram, and cardiac markers. The

most common ECG abnormality was sinus bradycardia seen in 9 (15%) patients, followed by sinus tachycardia in 6 (10%) and ST‐Tchanges in 5 (8.33%). Echocardiography was normal in 54 (90%) patients, systolic dysfunction was found in 4 (6.67%) patients, and pericardial effusion was found in 2 (3.33%) patients. Eight (13.33%) patients had abnormal creatine kinase‐muscle/brain values. The authors concluded that cardiac manifestations were common in dengue fever as they were seen in one third of their patients. Platelet counts less than 100,000/cu mm and a higher hematocrit were more likely to be associated with cardiac manifestations.[47]

Kalakutakar et al in 2019 described dengue fever as an acute febrile infectious disease, caused by any of four dengue serotypes. This study was undertaken to assess the cardiac manifestations in patients of dengue fever and was conducted in Karnataka Institute of Medical Sciences, Hubli. Dengue cases were confirmed serologically by detection of IgM antibodies. CKMB and Troponin I at admission were raised in 18% and 72% respectively. On ECG, 56% of the patients had normal rhythm, 15% had sinus bradycardia, 9% had sinus tachycardia, 10% had T wave changes, 3% had ST Changes, 2% had sinus bradycardia with T Wave Changes, 2% had sinus tachycardia with T wave changes and 1% had LVH and 2% had abnormal rhythm. The authors concluded that cardiac manifestations can cause significant morbidity in dengue patients and play an important role in management and assessing prognosis in this disease.[48]

Datta and Mitra in 2019 did a study with the objective of finding actual incidence of different cardiac manifestations of dengue fever in their tertiary care hospital. One hundred and twenty dengue patients were studied between January 2016 to December 2017. Routine biochemical parameters like complete haemogram, liver function tests, renal function tests and electrolytes were checked in all cases. ECG, echocardiography, Troponin T were evaluated in all patients and they were corroborated with clinical features like chest pain, dyspnoea, palpitation. Cardiac involvement in this study was seen in fifteen

patients (12.5%). Eight patients had bradyarrhythmias (6.6%). Asymptomatic sinus bradycardia was commonest (3.3%). All had normal recovery within two weeks. Four patients had left ventricular systolic dysfunction (ejection fraction 35% - 45%) and there was spontaneous recovery within three months. Two patients had pericardial effusion which resolved within two weeks. Transient 2:1 AV block and atrial fibrillation were observed in two cases. The authors specified that bradyarrhythmias were the most common manifestation in their study and it resolved spontaneously. [49]

Lee et al in 2020 undertook a retrospective analysis of dengue patients to assess risk factors for dengue hemorrhagic fever/ dengue shock syndrome and severe dengue. The study was done to elucidate the impact of co-existing co-morbidities like hypertension, chronic kidney disease, ischemic heart disease and glycemic control on clinical outcome of dengue in diabetic patients. DM2, hypertension and chronic kidney disease cause damage to vasculature in major organs and therefore cause dengue hemorrhagic fever/ dengue shock syndrome and severe dengue. However, their data suggested that dengue patients with DM2 and optimal sugar control were not at a higher risk of developing severe dengue.[50]

MATERIALS AND METHODS

This study was performed in the department of medicine, Dayanand Medical College and Hospital (DMCH) over a period of one year. All cases of dengue fever and those with cardiac complications were included in the study.In every case, a detailed clinical history and examination, with special attention to cardiac manifestations, was taken. Diagnosis of dengue fever was established by serological examination.

Cardiac manifestations were established by ECG, echo & cardiac biomarkers evaluation. From ECG, heart rate, rhythm abnormalities and signs of myocardial injury like ST-T segment changes were noted. On ECGSokolow-Lyon criteria were used for assessment of left ventricular hypertrophy. These included: R in v5 or v6 +S v1 or v2 more than 35 mm. R avL more than 11 mm.

Echocardiography revealed the presence of systolic dysfunction like regional or global wall motion abnormalities, ejection fraction, valvular abnormalities, left ventricular hypertrophy and pericardial effusion. 2D parasternal long axis view was used to determine the left ventricle end diastolic (LVED) and end systolic (LVES) dimensions to calculate ejection fraction. Left ventricular hypertrophy was calculated from the thickness of posterior wall of left ventricle by placing the cursors at tissue-blood interface just below the level of mitral valve as recommended by American society of echocardiography and European Association of cardiovascular imaging.

The cardiac biomarkers evaluated included CreatineKinase-MB, Troponin-T (TRT), D-dimer, myoglobin and lactate dehydrogenase (LDH). For CK-MB a value above 25 IU/L was considered elevated. For TRT a value above 0.4ng/ml was considered raised, for D-Dimer this value was above 250ng/ml, for myoglobin it was above 72ng/ml, for LDH it was above 280 U/L.

Diagnosis of myocardial injury was established either by ST-T segment changes on ECG or by reduced ejection fraction or wall motion abnormalities on echocardiography or by elevation of cardiac enzymes.

Patient mortality during hospital stay and during a 14-day period after discharge was noted.

The impact of cardiac manifestations on patient outcome was determined at the time of discharge and after a period of 14 days post discharge.

All patient data was collected on a specially designed proforma. Written informed consent was obtained from all patients before enrolling them in this study. SPSS software was used for data analysis.

INCLUSION CRITERIA

1. All patients of dengue fever and those with cardiac manifestations, of adult age, and of any gender, admitted in DMCH were included in the study.

2. Only those patients were included in the study whose diagnosis of dengue fever was confirmed by serological examination.

EXCLUSION CRITERIA

Patients with pre-existing cardiac diseases were excluded on the basis of history.

Patients who had fever due to causes other than dengue fever were also excluded.

SOURCE OF DATA

Data was sourced from the indoor patients admitted in DMCH.

STATISTICAL ANALYSIS

Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate.For comparing categorical data, Chi square ($\chi 2$) test was performed and exact test was used when the expected frequency is less than 5.A probability value (*p* value)less than 0.05 was considered statistically significant.All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21version(SPSS Inc., Chicago, IL, USA)statistical program for Microsoft Windows.

OBSERVATIONS & RESULTS

TABLE 1. AGE DISTRIBUTION

Age Groups (in years)	No. of Patients	%age
10 - 29	69	24.56 %
30 - 49	114	40.57 %
50 - 69	80	28.47 %
>69	18	6.41 %
Total	**281**	**100.00 %**
Mean±SD	41.91±16.13	

The maximum number of patients (114) was between the age group of 30-49 years comprising 40.57% of all cases. In the older age group of more than 69 years, only 18 patients were present comprising 6.4% of total cases.

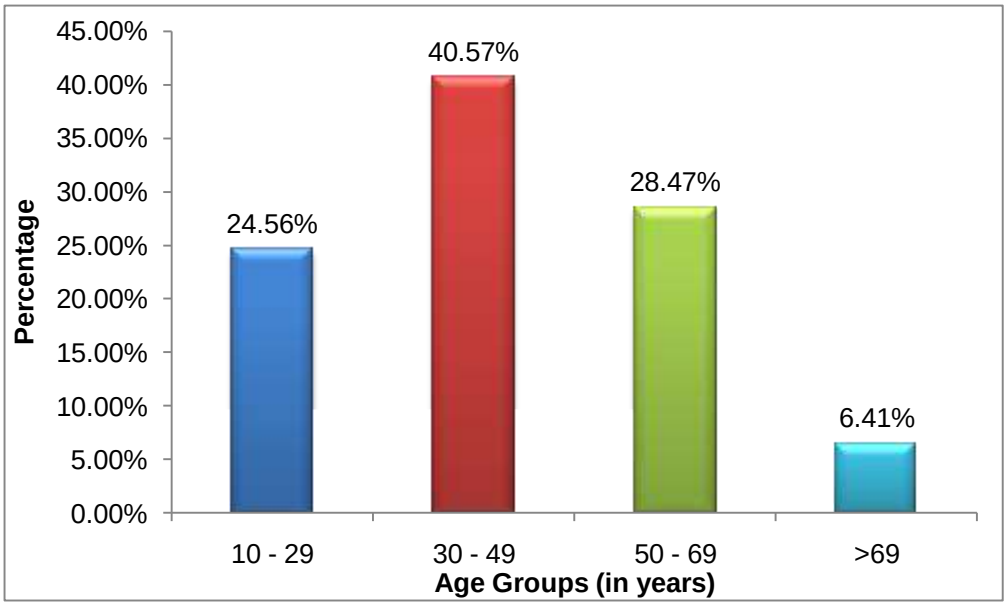

FIGURE 1. AGE DISTRIBUTION

TABLE 2. GENDER DISTRIBUTION

Gender	No. of Patients	%age
Male	200	71.17 %
Female	81	28.83 %
Total	**281**	**100.00 %**

Out of 281 patients, 200 were male comprising 71.17% of cases. Only 81 patients comprising 28.83% of all cases were female.

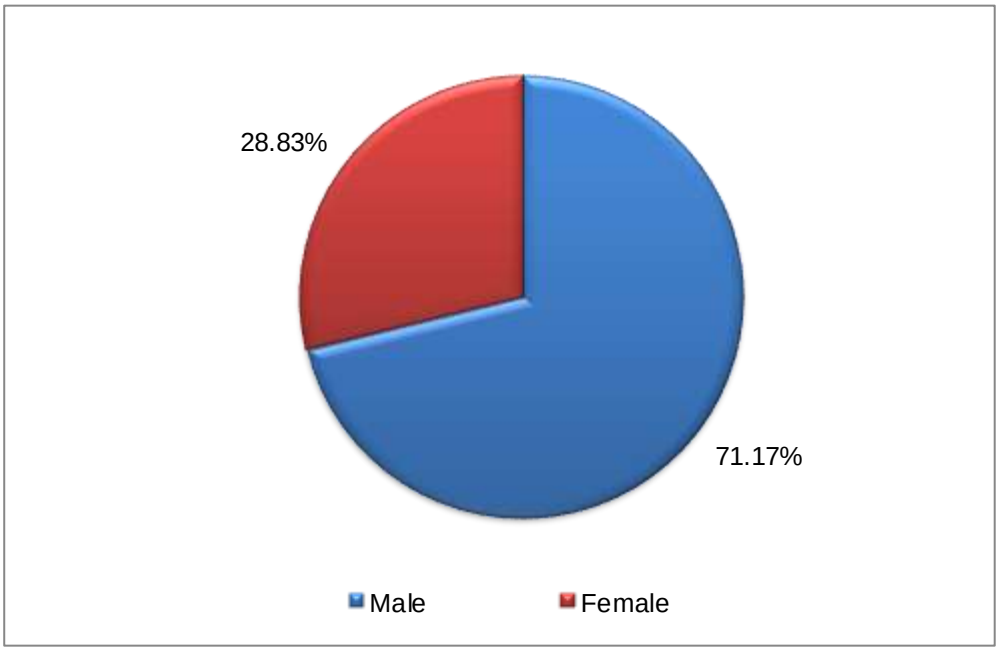

FIGURE 2. GENDER DISTRIBUTION

TABLE 3. TLC DISTRIBUTION

TLC	No. of Patients	Percentage
<2000	**12**	**4.3**
2000-4500	**80**	**28.4**
4501-11000	**166**	**59**
>11000	**23**	**8.3**
TOTAL	**281**	**100**

Twelve patients had TLC of less than 2000/cumm (4.3%). 80 patients had count between 2000-4500/cumm (28.4%). 166 patients had normal counts between 4501-11,000/cumm (59%). 23 patients had a high TLC of more than 11000/cumm (8.3%).

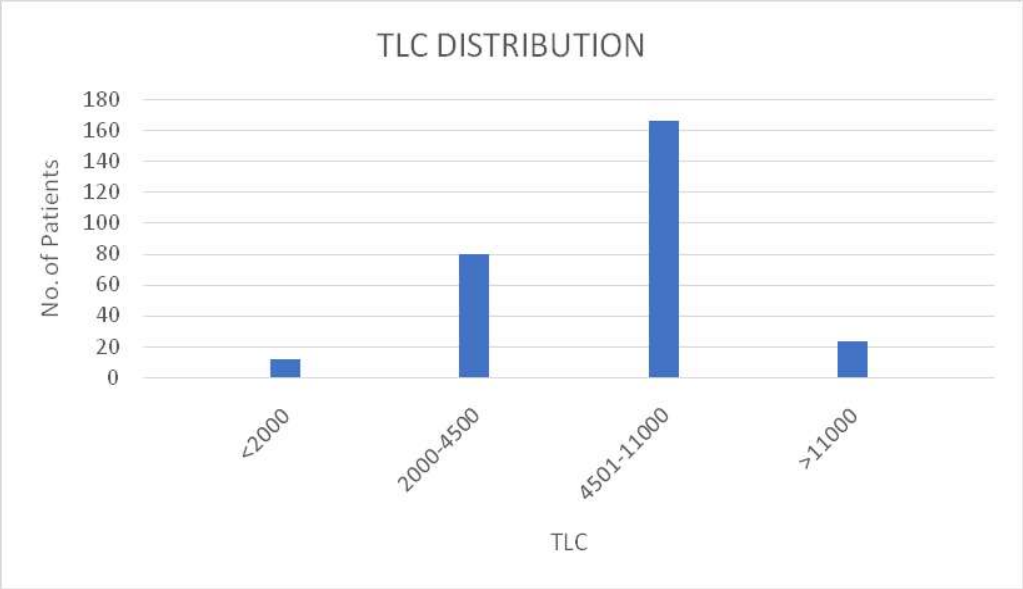

FIGURE 3. TLC DISTRIBUTION

TABLE 4. DLC NEUTROPHILS DISTRIBUTION

DLC Neutrophils	No. of Patients	%age
<40	35	12.46 %
40 - <60	122	43.42 %
>60	124	44.13 %
TOTAL	**281**	**100.00 %**

Neutropenia was observed in 35 patients (12.46%). The count was raised in 124 patients (44.13%). Neutrophils were normal in 122 patients (43.42%)

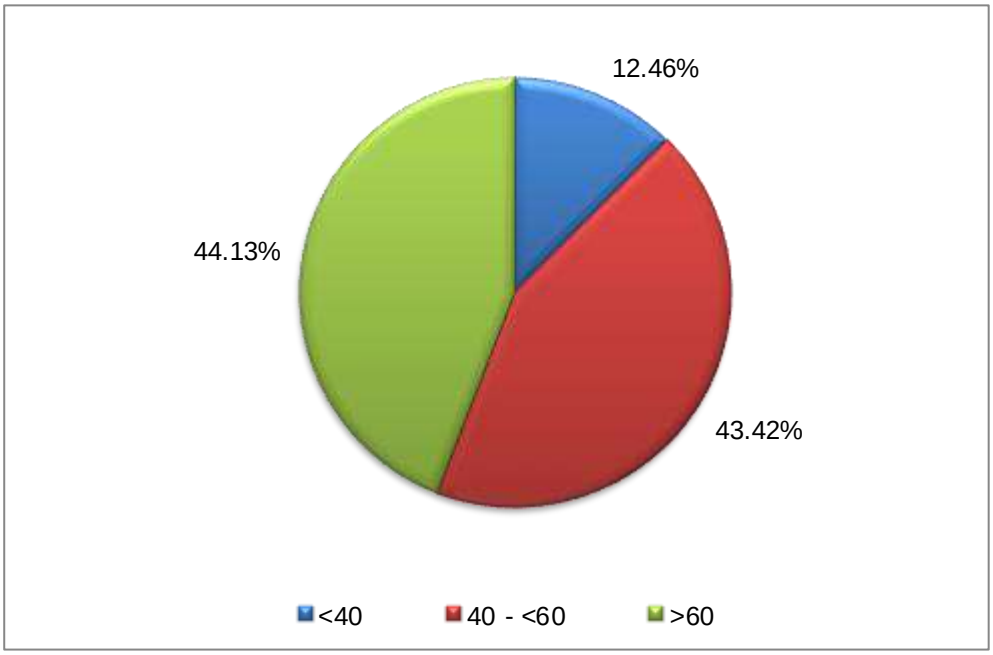

FIGURE 4. DLC NEUTROPHILS DISTRIBUTION

TABLE 5. DLC LYMPHOCYTE DISTRIBUTION

DLC Lymphocyte	No. of Patients	%age
<20	35	12.46 %
20 - <40	158	56.23 %
>40	88	31.32 %
TOTAL	**281**	**100.00 %**

Lymphopenia was seen in 35 patients (12.46%). Lymphocytosis was seen in 88 patients (31.32%). 158 patients showed normal lymphocyte count (56.23%)

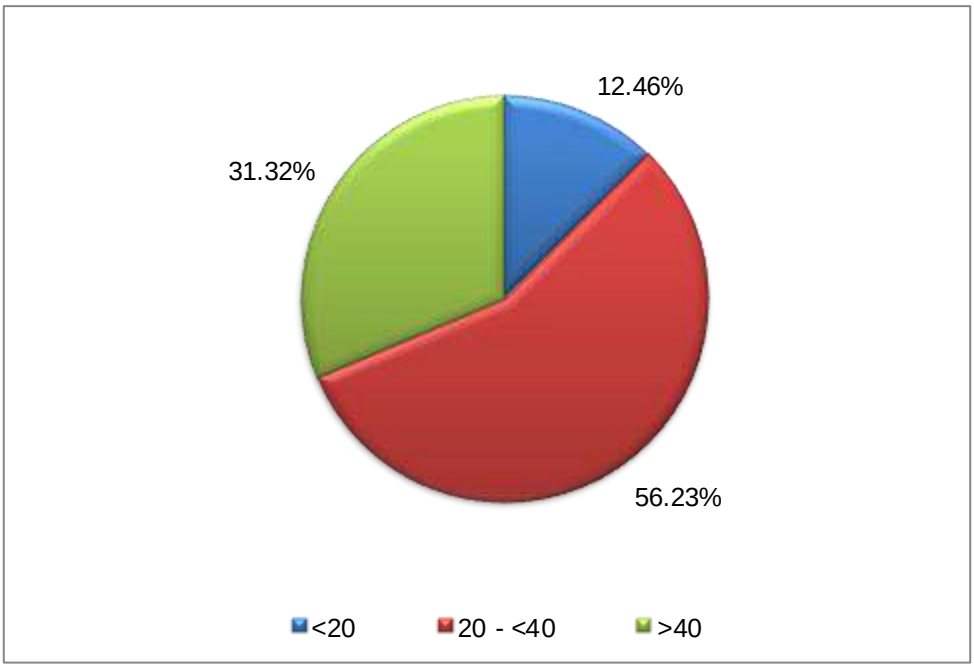

FIGURE 5. DLC LYMPHOCYTE DISTRIBUTION

TABLE 6. PLATELET COUNT DISTRIBUTION

PLT GROUP	No. of cases	Percentage
< 20,000	30	10.7
20000-50,000	102	36.3
50,001-150,000	121	43.0
>150,000	28	10.0
Total	**281**	**100.0**

253 patients (90%) showed thrombocytopenia. 30 patients (10.7) showed a platelet count below 20,000/cumm. 102 patients (36.3%) had a count between 20,000-50,000/cumm. In 121 patients (43%) the platelet count was between 50001-150,000/cumm. A normal platelet count above150,000 was seen in only 28 patients (10%).

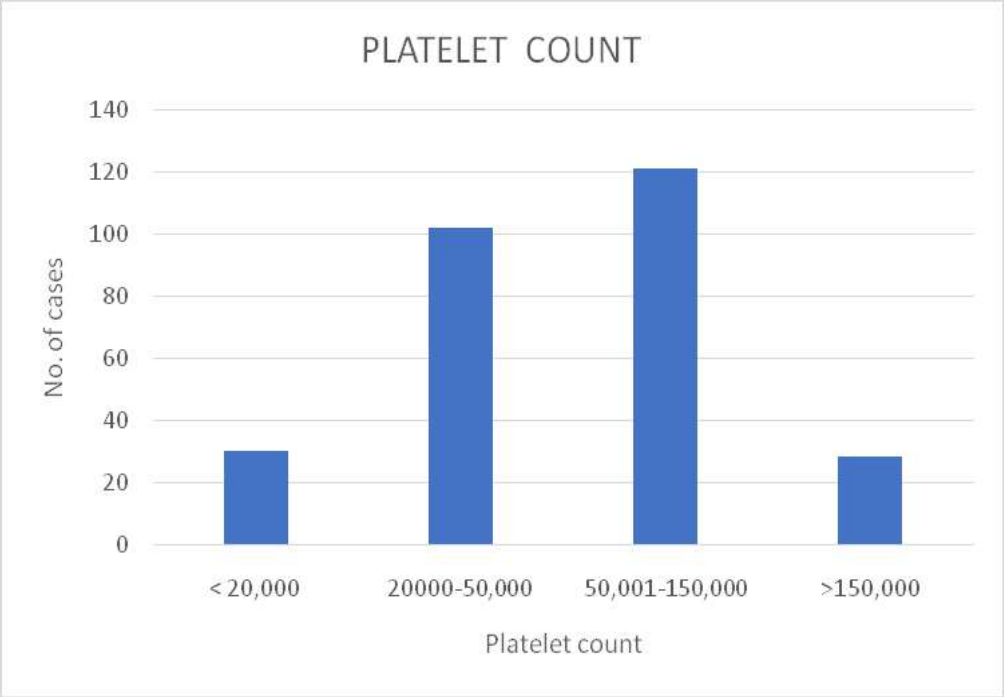

FIGURE 6. PLATELET COUNT DISTRIBUTION

TABLE 7. DISTRIBUTION OF CREATININE CLEARANCE (CRCL)

Crcl	No. of Patient	%age
< 15	3	1.07%
15-30	5	1.78%
31-60	8	2.84%
60-89	2	0.72%
> 90	263	93.59%

18 patients had a low creatinine clearance indicative of chronic kidney disease. They comprised 6.40% of the total cases.

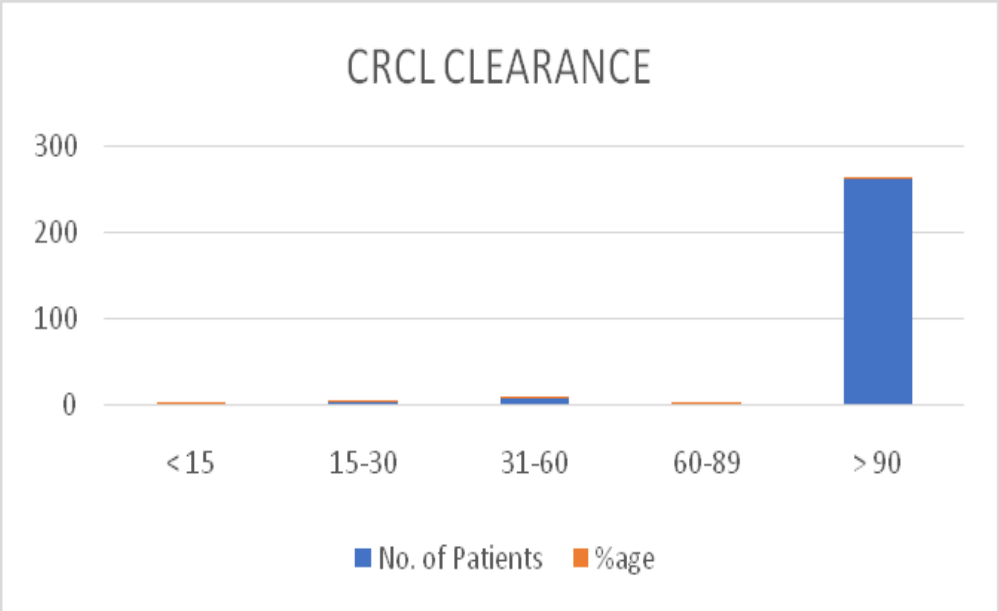

FIGURE 7. DISTRIBUTION OF CREATININE CLEARANCE (CRCL)

TABLE 8. SGOT DISTRIBUTION ACCORDING TO GENDER

SGOT	Male		Female	
	No. of Patients	%age	No. of Patients	%age
8-44	24	12.00 %	6	7.41 %
>44	176	88.00 %	75	92.59 %
TOTAL	**200**	**100.00 %**	**81**	**100.00 %**

SGOT was elevated in 176 male patients (88%) and 75 female patients (92.59%)

FIGURE 8. SGOT DISTRIBUTION ACCORDING TO GENDER

TABLE 9. SGPT DISTRIBUTION ACCORDING TO GENDER

SGPT	Male		Female	
	No. of Patients	%age	No. of Patients	%age
7-55	72	36.00 %	28	34.57 %
>55	128	64.00 %	53	65.43 %
TOTAL	**200**	**100.00 %**	**81**	**100.00 %**

SGPT was elevated in 128 male patients (64%) and 53 female patients (65.43%)

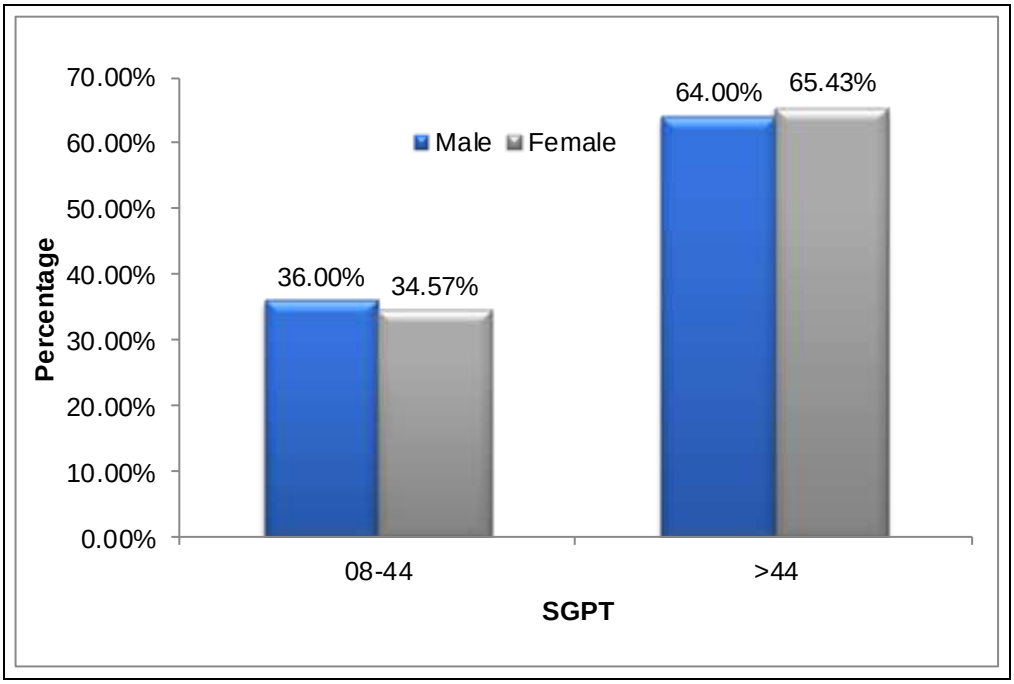

FIGURE 9. SGPT DISTRIBUTION ACCORDING TO GENDER

TABLE 10. RATIO OF SGOT/SGPT

OT/PT GROUP	No. of cases	Percentage
< 1	**33**	**11.7**
> 1	**248**	**88.3**
Total	**281**	**100.0**

The OT/PT ratio was less than 1 in 33 cases (11.7%). It was more than 1 in 248 cases (88.3%).

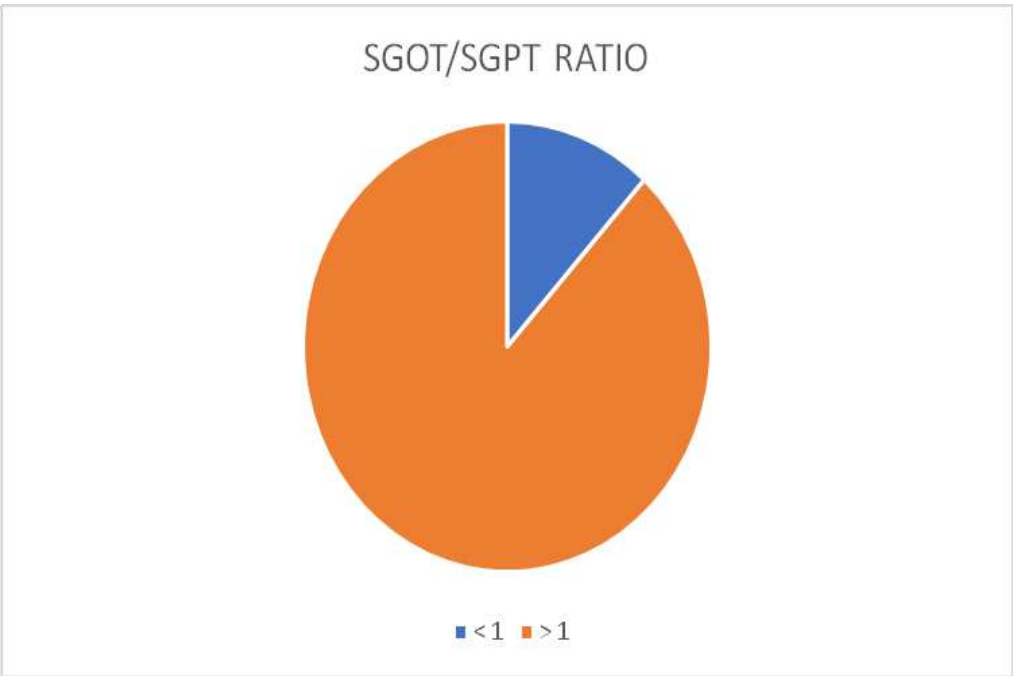

FIGURE 10. RATIO OF SGOT/SGPT

TABLE 11. CHEST X-RAY (CXR) FINDINGS

CXR findings	No. of Patients	%age
Pleural Effusion	56	19.93%
B/l infiltration	37	13.16%
cardiomegaly	3	1.07%
Normal	185	65.84%

Pleural effusion was detected in 56 patients (19.93%). Bilateral infiltration was observed in 37 patients (13.16%). Cardiomegaly was observed in 3 patients (1.07%). CXR was normal in 185 patients (65.84%).

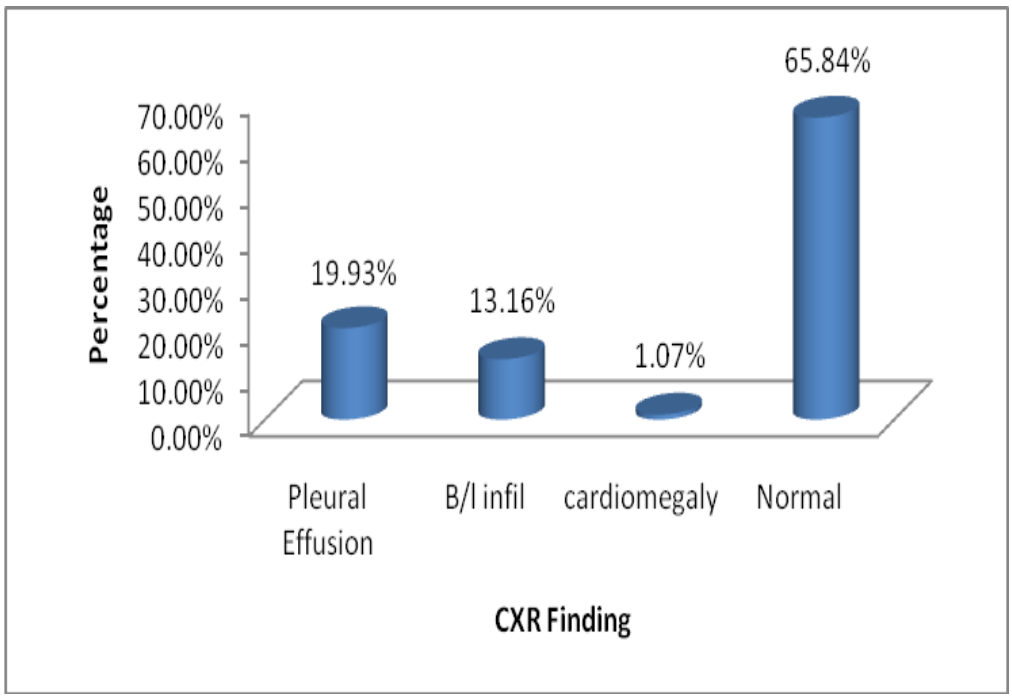

FIGURE 11. CHEST X-RAY (CXR) FINDINGS

TABLE 12. HEART RATE DURING HOSPITAL STAY

HR	No. of Patients	%age
≤ 40	3	1.07%
41-60	132	46.98%
61-100	144	51.24%
> 100	2	0.71%
Total	281	100.0%

135 patients showed bradycardia with a heart rate of less than 60. Heart rate was less than 40 in 3 patients (1.07%). 2 patients showed tachycardia with heart rate above 100. These comprised 0.71% cases.

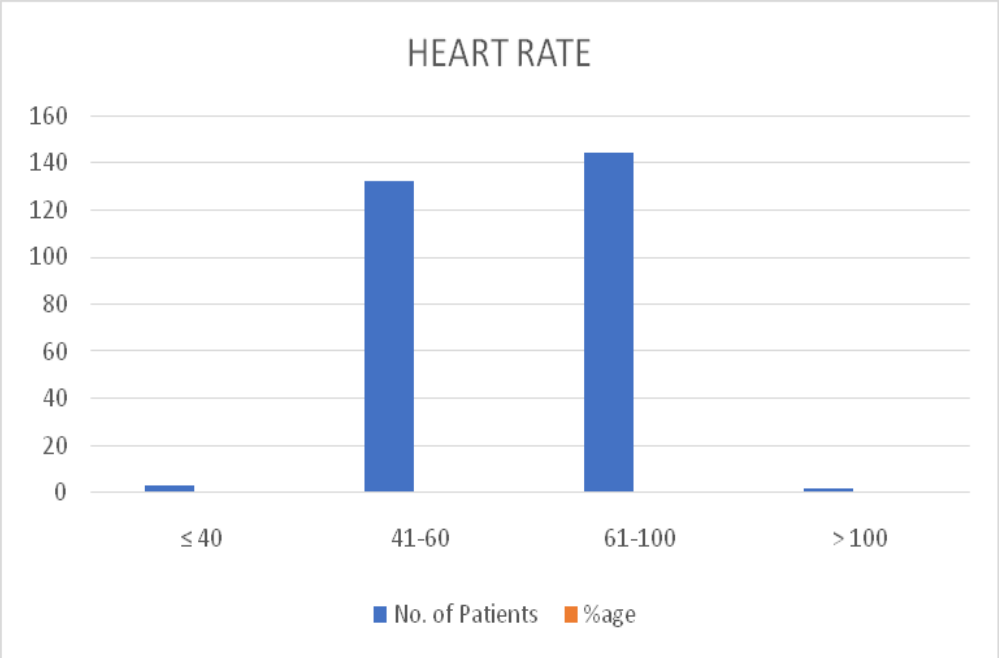

FIGURE 12. HEART RATE DURING HOSPITAL STAY

TABLE 13. ECTOPICS DISTRIBUTION

Ectopics	No. of Patients	%age
Present	3	1.07 %
Absent	278	98.93 %
TOTAL	**281**	**100.00 %**

Ectopics were present in 3 cases comprising 1.07% of all cases.

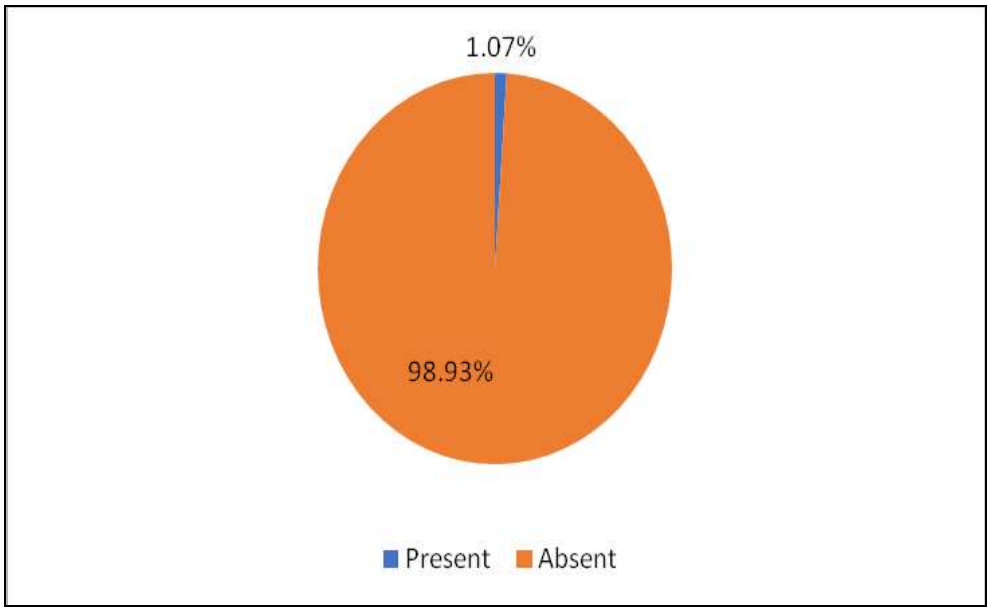

FIGURE 13. ECTOPICS DISTRIBUTION

TABLE 14. BRADYCARDIA DISTRIBUTION

Bradycardia	No. of Patients	%age
Present	135	48.04 %
Absent	146	51.96 %
TOTAL	**281**	**100.00 %**

Bradycardia was observed in 135 patients comprising 48.04% of all cases.

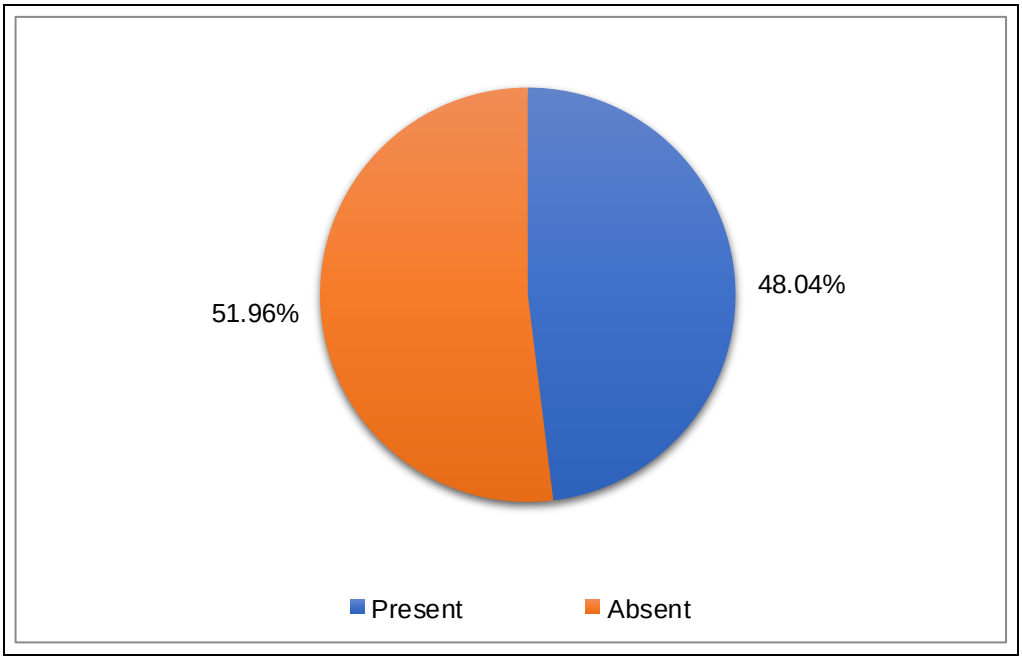

FIGURE 14. BRADYCARDIA DISTRIBUTION

TABLE 15.TACHYCARDIA DISTRIBUTION

Tachycardia	No. of Patients	%age
Present	2	0.71%
Absent	279	99.29%
TOTAL	**281**	**100.00 %**

Tachycardia was observed in 2 patients comprising 0.71% of all cases.

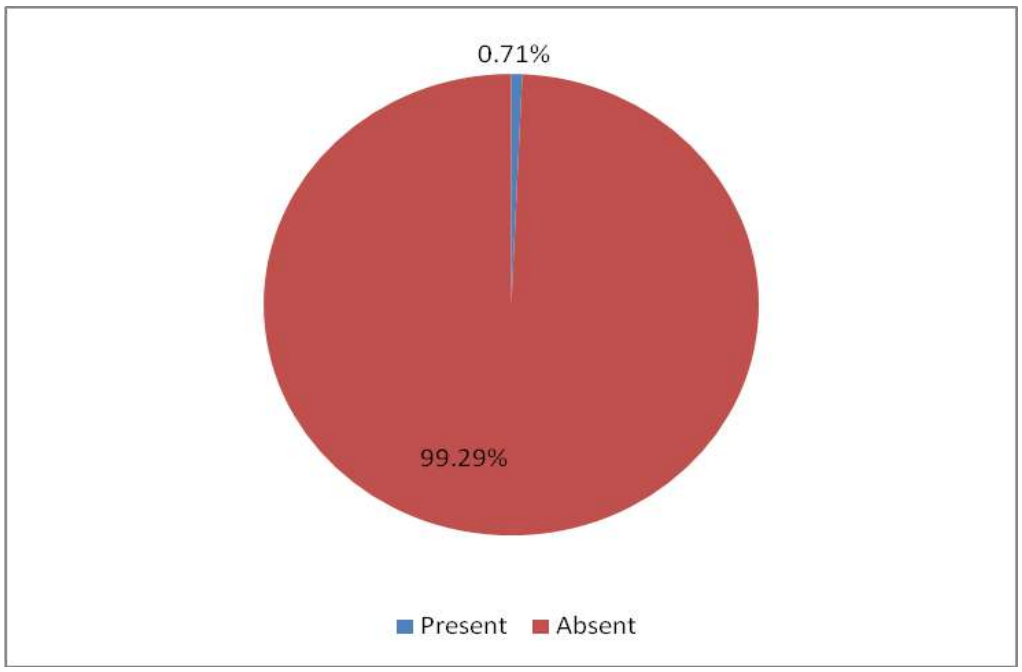

FIGURE 15. TACHYCARDIA DISTRIBUTION

TABLE 16. LEFT VENTRICULAR EJECTION FRACTION (LVEF) DISTRIBUTION

LVEF	No. of Patients	%age
<35	3	1.07%
36-50	4	1.42%
>51	274	97.51%
TOTAL	**281**	**100.00 %**

3 patients had an ejection fraction of less than 35% and 4 patients had an ejection fraction of less than 50%. So, ejection fraction was reduced in a total of 7 patients.

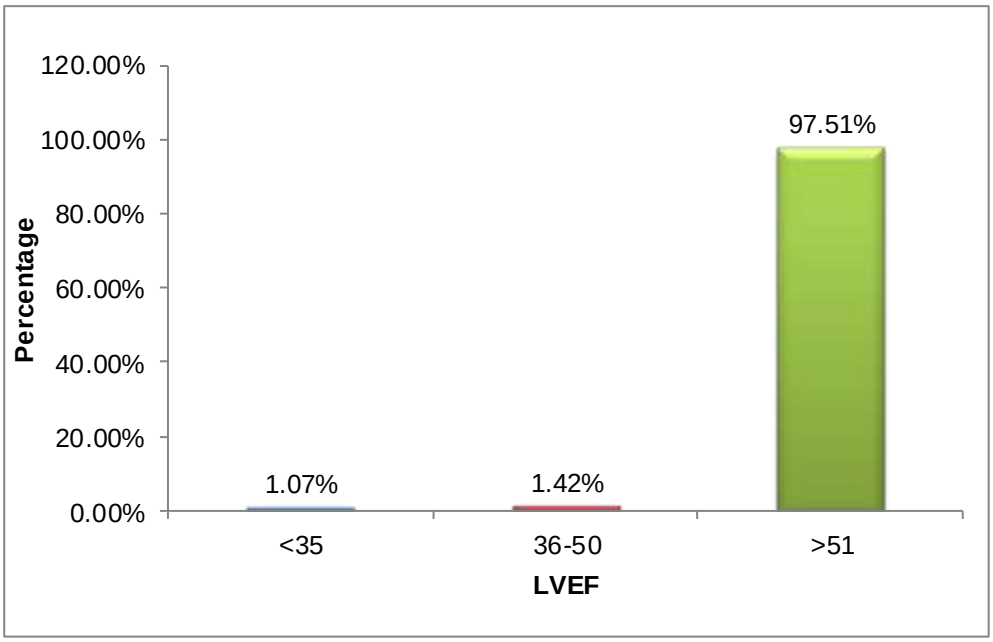

FIGURE 16. LEFT VENTRICULAR EJECTION FRACTION (LVEF) DISTRIBUTION

TABLE 17. CREATINE KINASE-MB (CKMB) DISTRIBUTION

CKMB	No. of Patients	%age
5-25	276	98.22 %
>25	5	1.78%
TOTAL	**281**	**100.00 %**

5 Patients (1.78%) had elevated CKMB.

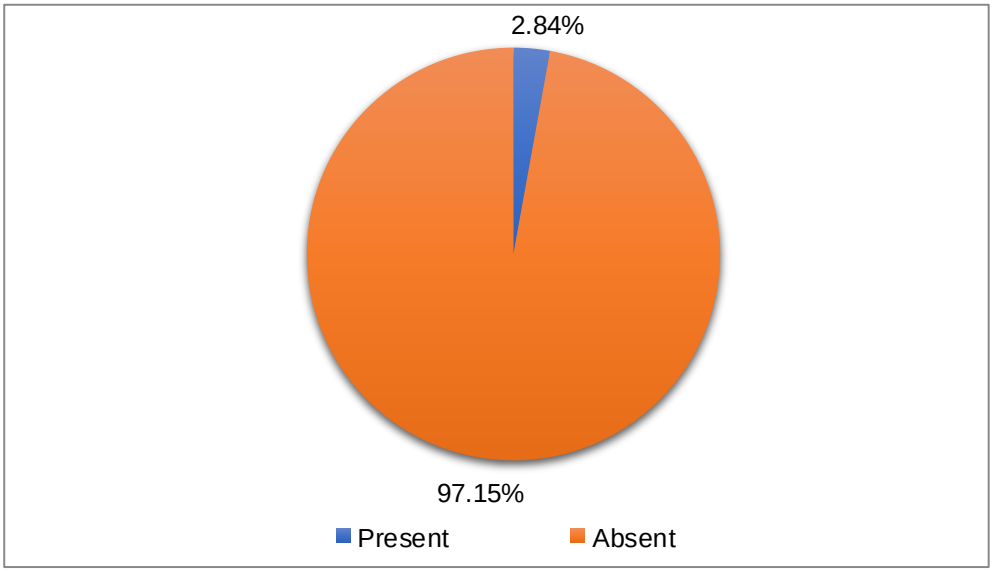

FIGURE 17. CREATINE KINASE-MB (CKMB) DISTRIBUTION

TABLE-18. TROPONIN-T (TRT) DISTRIBUTION

TRT	No. of Patients	%age
0 - 0.4	269	95.72 %
>0.04	12	4.27%
TOTAL	**281**	**100.00 %**

In 12 patients (4.27%) troponin-T was found to be elevated.

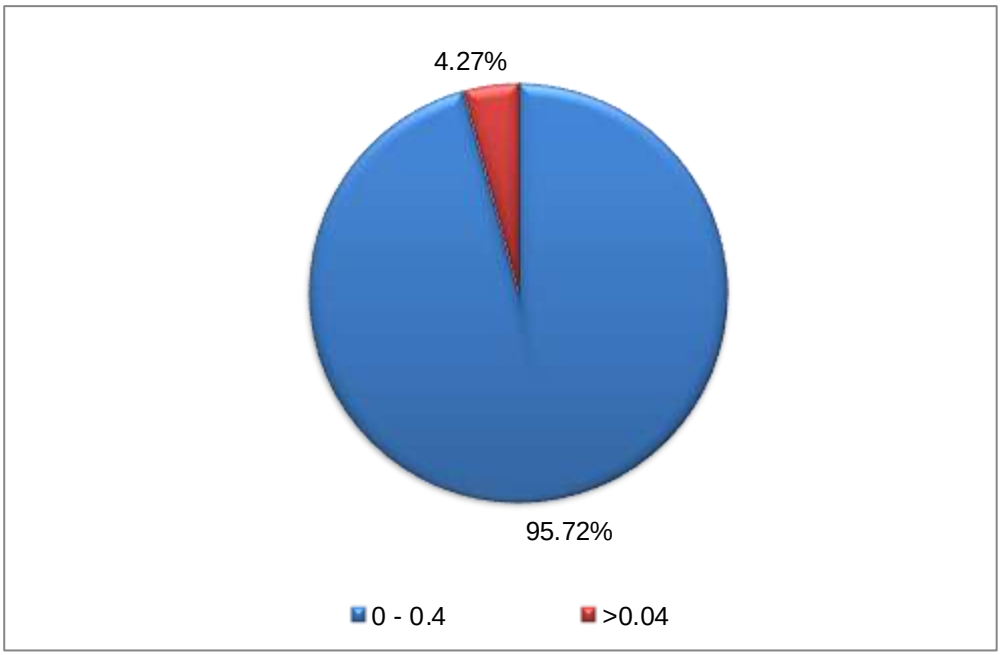

FIGURE 18. TROPONIN-T (TRT) DISTRIBUTION

TABLE 19. D-DIMER DISTRIBUTION

D-DIMER	No. of Patients	%age
<250	233	82.92 %
>250	48	17.08 %
TOTAL	**281**	**100.00 %**

D-Dimer was elevated in 48 patients comprising 17.08% cases.

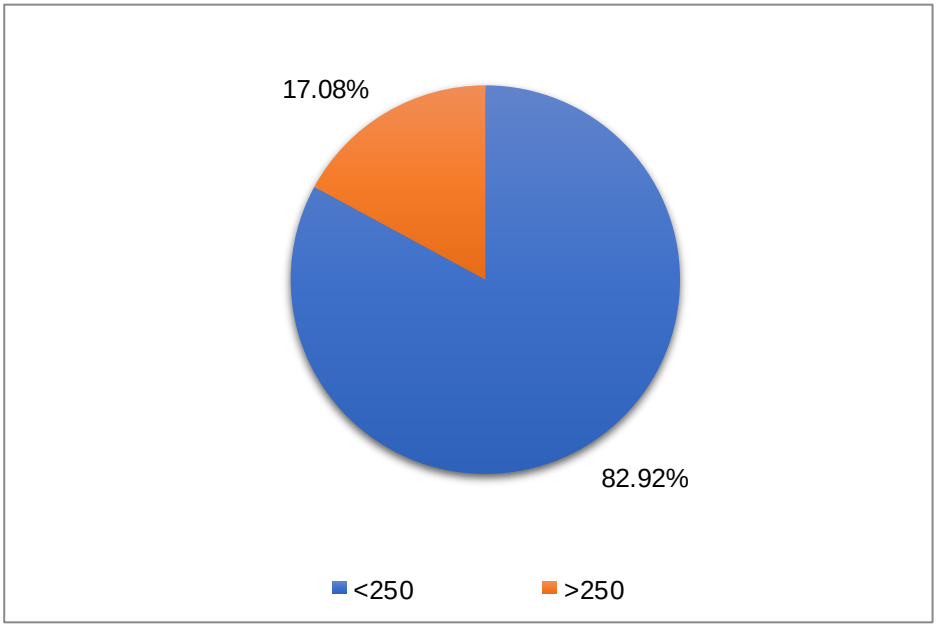

FIGURE 19. D-DIMER DISTRIBUTION

TABLE 20. MYOGLOBIN DISTRIBUTION

Myoglobin	No. of Patients	%age
25-72	240	85.41 %
>72	41	14.59 %
TOTAL	**281**	**100.00 %**

Myoglobin was elevated in 41 patients comprising 14.59% Cases.

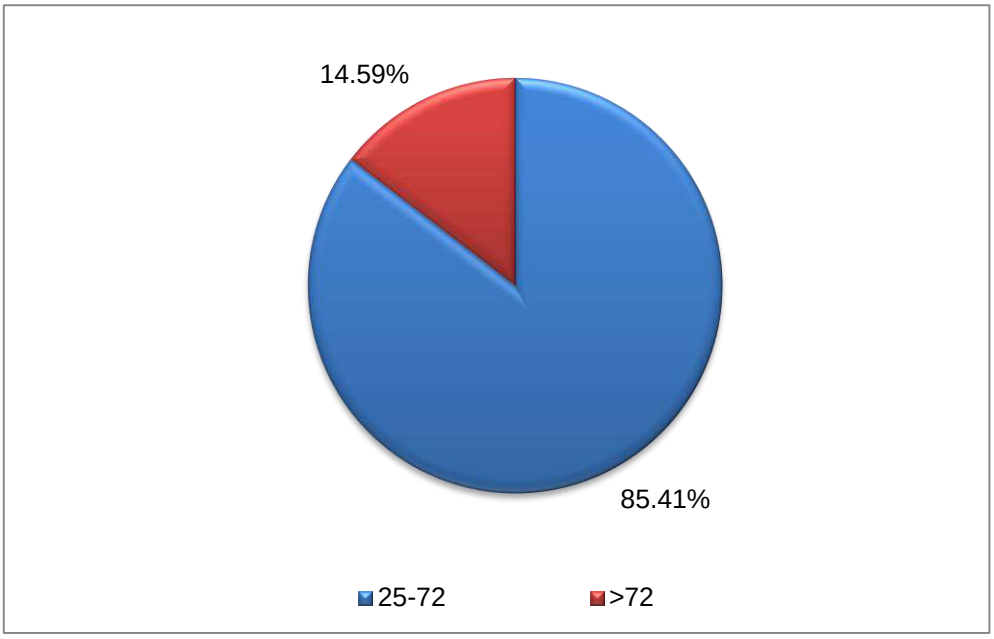

FIGURE 20. MYOGLOBIN DISTRIBUTION

TABLE 21. LACTATE DEHYDROGENASE (LDH) DISTRIBUTION

LDH	No. of Patients	%age
<140	8	2.85 %
140-280	242	86.12 %
>281	31	11.03 %
TOTAL	**281**	**100.00 %**

LDH was elevated in 31 patients comprising 11.03% cases.

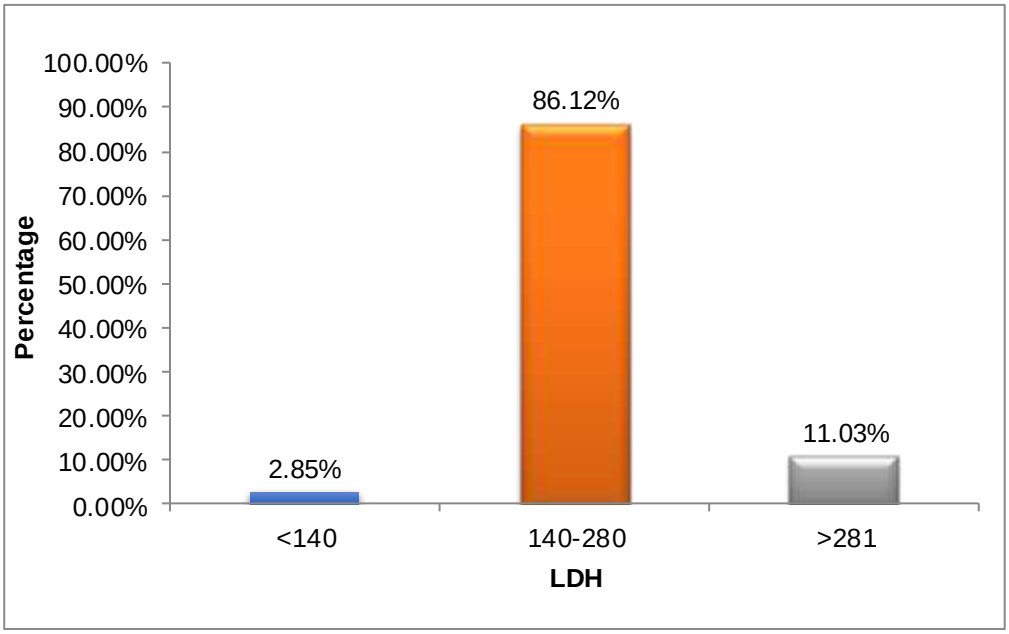

FIGURE 21. LACTATE DEHYDROGENASE (LDH) DISTRIBUTION

TABLE 22. CARDIAC MANIFESTATION DISTRIBUTION

Cardiac Manifestation	No. of Patients	%age
BBB	18	6.41%
Bradycardia	135	48.04%
Ectopics	3	1.07%
LVH	1	0.36%
MR	1	0.36%
Myocardial Injury	88	31.32%
Normal	97	34.52%
Mortality	2	0.71%
Tachycardia	2	0.71%

Note: some patients had more than one cardiac manifestation.

During hospital stay, the distribution of cardiac manifestations was as depicted in the table. There were 18 patients of bundle branch block (BBB). 135 cases had bradycardia which was the most common cardiac manifestation. 3 cases had ectopics, one had left ventricular hypertrophy (LVH), one had mitral regurgitation (MR), 88 cases had myocardial injury and 2 had tachycardia. Two patients succumbed to the disease during the course of hospital stay. Both these patients had markedly reduced ejection fractions (20% & 25% along with global hypokinesia).

As few patients had more than one cardiac manifestation, so the number of cardiac manifestations is more than the number of patients included in the study.

FIGURE 22. CARDIAC MANIFESTATION DISTRIBUTION

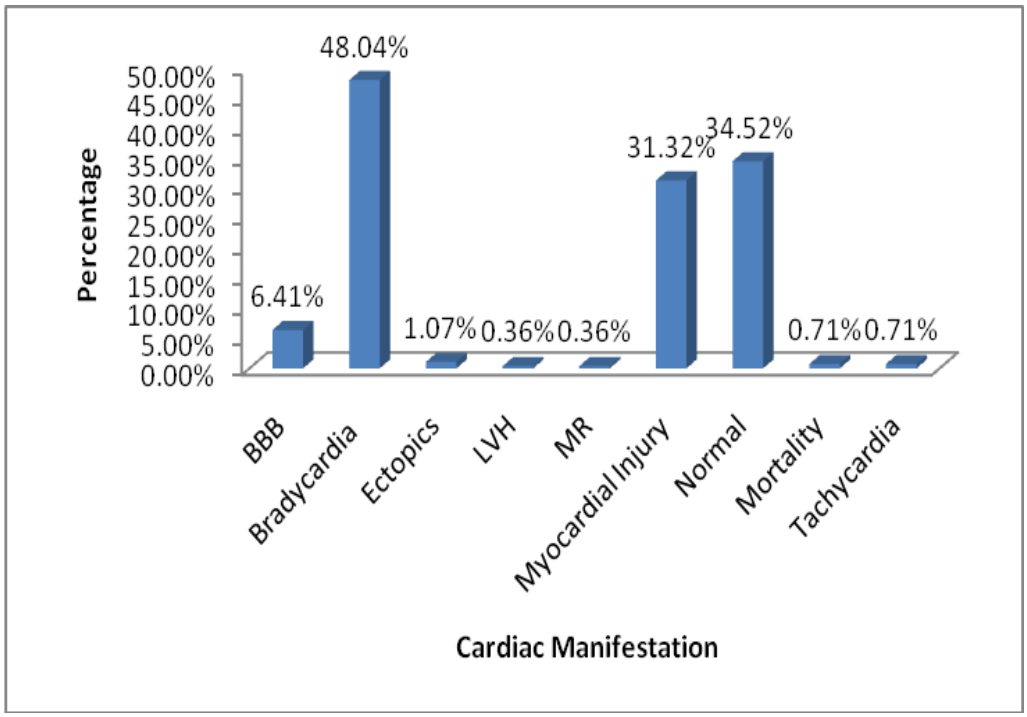

TABLE 23. OUTCOME OF CARDIAC MANIFESTATIONS

Cardiac Manifestation	No. of Patients	At the time of discharge	14 Days Outcome
BBB	18	5	0
Bradycardia	135	0	0
Ectopics	3	0	0
LVH	1	1	1
MR	1	1	1
Myocardial Injury	88	0	0
Normal	97	272	277
Mortality	2	0	0
Tachycardia	**2**	0	0
p value		**<0.001**	

Fisher's exact

Note: This table depicts the cardiac manifestations during hospital stay, at the time of discharge and 14 days post-discharge. At the time of discharge, 5 patients showed residual bundle branch block which resolved after a period of 14 days.

One patient showed left ventricular hypertrophy and one patient showed mitral regurgitation on echo.

After a period of 14 days, only one patient showed left ventricular hypertrophy and one patient showed mitral regurgitation on echo. The resolution of most cardiac manifestations indicates the reversible nature of cardiac injury in dengue patients.

FIGURE 23. OUTCOME OF CARDIAC MANIFESTATIONS

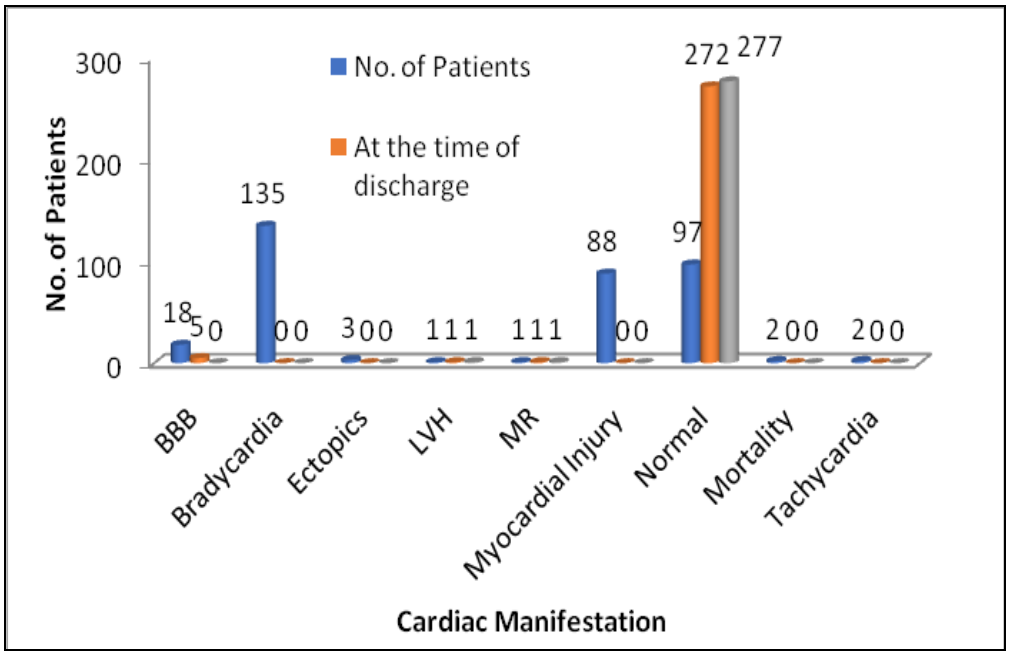

TABLE 24. CO-RELATION BETWEEN TLC AND CARDIAC MANIFESTATIONS

TLC	Cardiac Manifestation			
	Yes		No	
	Number	Percentage	Number	Percentage
<4500	42	52.5%	38	47.5%
4500 - <11000	117	70.48%	49	29.52%
>11000	25	71.43%	10	28.57%
Total	184	65.48	97	34.52
P value	**0.015**			

Chi square Test

No significant co-relation was observed between leucocyte count and cardiac manifestations in dengue patients.

TABLE 25. CO-RELATION BETWEEN PLATELET COUNT AND CARDIAC MANIFESTATIONS

Platelet Count (cum^2)	Cardiac Manifestation			
	Yes		No	
	Number	Percentage	Number	Percentage
<150	162	64.80%	88	35.20%
>150	22	70.97	9	29.03%
Total	**184**	**65.48**	**97**	**34.52**
P value		**0.496**		

Chi Square test

No significant co-relation was observed between thrombocytopenia and cardiac manifestations in dengue patients.

TABLE 26. CO-RELATION BETWEEN CRITICAL PLATELET COUNT AND CARDIAC MANIFESTATIONS

Platelet Count (cum^2)	Cardiac Manifestation			
	Yes		No	
	No. of Patients	%age	No. of Patients	%age
<20000	16	53.33%	14	46.67%
20000 - 50000	67	65.69%	35	34.31%
50000-150000	82	67.77%	39	32.23%
> 150000	19	67.86%	9	32.14%
Total	184	65.48%	97	34.52%
P value	0.512			

Fisher Exact Test

No significant co-relation was observed in critically low platelet count and cardiac manifestations in dengue patients.

TABLE 27. CO-RELATION BETWEEN CO-MORBIDITIES AND CARDIAC MANIFESTATIONS

Cardiac Manifestation

Co morbidities	Yes		No	
	Number	**Percentage**	**Number**	**Percentage**
CKD	16	88.89%	2	11.11%
CLD	4	40.00%	6	60.00%
DM2	32	86.49%	5	13.51%
HTN	35	72.92%	13	27.08%
Normal	97	57.74%	71	42.26%
Total	**184**	**65.48%**	**97**	**34.52%**
P value	**<0.001**			

Fisher Exact Test

A significant co-relation was observed between cardiac manifestations and co-morbidities like chronic kidney disease (CKD), diabetes (DM2) & hypertension (HTN).

DISCUSSION

The present study was conducted on 281 patients of dengue fever admitted in DMCH Ludhiana.

Age of the Patient

In the present study the maximum number of patients were in the age group 30-49 years with the mean age of 41 years. Only 6.4% of the patients were aged more than 69 years.

This is in consonance with the study of Gupta & Gadpayle 2010, who found a mean age of 30.1 years.

In their study of 2013, Miranda et al also found a mean age of 32 years.

Gender Distribution

In the present study, overwhelming majority of patients were male. Males comprised 71.1% of cases while females comprised 28.8% cases.

In the study by Gupta & Gadpayle in 2010, 64.28% cases were males and 35.7% cases were females.

The study by Miranda et al in 2013 found 81.25% cases to be male.

In the study by Tarique et al in 2013, males were 81% and females were 19%

These studies corroborate with the present study in gender distribution of cases.

TLC and DLC Count

Leucopenia was observed in 32.7% of cases in the present study. Majority of the patients (59%) showed a normal leucocyte count. In our study neutropenia was observed in 35 patients (12.46%). Neutrophils were normal in 122 patients (43.42%). Neutrophil count was raised in 124 patients (44.13%). Lymphopenia was observed in 35 patients (12.46%). Lymphocyte count was normal in 158 patients (56.23%). Lymphocytosis was observed in 88 patients (31.32%).

The mean TLC in the study by Miranda et al in 2013 was also within normal range. Mean TLC was 4907/cumm.

Azin et al in 2012 found leucocytosis in the first few days of the disease followed by leucopenia. Similarly, there was a decrease in lymphocytes in the early days of disease followed by lymphocytosis.

Platelet Count

Thrombocytopenia was observed in 88.97% of the cases in the present study. 10.7% patients in our study showed critical thrombocytopenia with a cell count below 20,000/cumm.

Miranda et al in 2013 found thrombocytopenia in 80.3% cases.

Gupta & Gadpayle 2010 found thrombocytopenia in all their patients (100%).

These studies confirm the high incidence of thrombocytopenia in dengue cases and corroborate with the present study.

CORRELATION OF CARDIAC MANIFESTATIONS WITH TLC, PLATELET COUNT AND CO-MORBID CONDITIONS

In the present study, there was no significant co-relation between total leucocyte count and cardiac manifestations. Similarly, the platelet count did not co-relate with cardiac manifestations. However, a significant co-relation was observed between patients of chronic kidney disease, diabetes and hypertensive patients who showed a higher percentage of cardiac manifestations.

Omar et al in 2013 found increased mortality in dengue patients with co-morbidities like diabetes mellitus.

Lee et al in 2020 also established that dengue patients with co-morbidities like diabetes mellitus (DM2), hypertension and chronic kidney disease have a higher risk of developing severe dengue.

Both these studies corroborate with the present study.

Serum Transaminases

In our study SGOT was elevated in 88% of male patients and in 92.59% of female patients.SGPT was elevated in 64% of male patients and 65.43% of female patients.

Miranda et al in 2013 found raised SGOT in 82.60% of cases and raised SGPT in 82.35% cases

Nimmagadda et al in 2014 found raised SGOT in 92.7% cases and raised SGPT in 85.3% cases.

Both these studies co-relate with the present study.

Chest X-Ray Abnormalities

In the present study chest X-ray found pleural effusion in 56 cases (19.93%). Bilateral infiltration was found in 37 cases (13.16%). Cardiomegaly was found in 3 cases (1.07%).

Gupta & Gadpayle in 2010 found pleural effusion in 14% cases. This study corelates with the present study.

Wang et al in 2007 detected infiltration in 23.3% cases and pleural effusion in 25% cases. Effusion was predominantly right sided. Infiltration was bilateral in 45% cases.

Bradycardia

In the present study bradycardia was the most common cardiac manifestation and was found in 135 patients (48.04%).

Chopra in 2018 found bradycardia in 33% of patients with dengue fever. The incidence increased to 45% when patients with conduction defects were also included.

Tarique et al in 2013 found bradycardia in 21.3% of their cases of dengue fever.

Both these studies are in consonance with the present study.

Gupta & Gadpayle found bradycardia in 14.28% cases. This low incidence of bradycardia is ascribed to the fact that none of their patients had overt myocardial injury and study was confined to subclinical cardiac involvement in dengue.

Tachycardia

In the present study tachycardia was observed in 2 cases (0.71%). This was self- limiting and resolved by the time of discharge.

Salgado et al in 2010 found tachycardia in 1.96% cases. Their study co-relates with the present study.

Tarique et al in 2013 found tachycardia in 3.2% of cases. In their study also the incidence of tachycardia was low. Tachycardia was found to be self-limiting and was ascribed to high fever.

Ectopics

In the present study ectopics were present in 3 patients comprising 1.07% of all cases.

La -Orkhun et al in 2011 reported atrial ectopics in 11% cases of dengue fever in the convalescent stage. Their higher incidence is ascribed to the fact that they had included only children in their study.

Cardiac Enzymes

In the present study, CK-MB was elevated in 1.78%, TRT in 4.27%, D-Dimer was elevated in 17.08% cases, Myoglobin in 14.59% and LDH in 11.03% cases. Elevation of these enzymes was taken as indicator of myocardial injury.

Salgado et al in 2010 found that CPK-MB was raised in 5.88% of dengue cases.

Miranda et al in 2013 found elevated cardiac biomarkers in 15% of dengue patients which corroborates with our study.

Kirawittaya et al in 2015 found that Trop-T was elevated in 14.5% of patients with dengue hemorrhagic fever and in 5% of cases with dengue fever. This study is also in consonance with the present study.

Left ventricular Ejection Fraction

In the present study ejection fraction was less than 50% in 7 patients (2.49%). 3 patients had an ejection fraction of less than 35% comprising 1.07% of all cases. 4 patients had a reduced ejection fraction between 36-50% comprising 1.42% cases. Two patients who had ejection fraction of 20% and 25% succumbed to the disease during their hospital stay. In all other cases the ejection fraction improved to normal levels at the time of discharge, indicating that myocardial depression was self-limiting and transient in most of the cases.

Salgado in 2010 found reduced ejection fraction in 1% cases. This result is similar to our study.

In 2003 Khongphatthanayothin et al found reduced ejection fraction of less than 50% in 6.7% cases. The ejection fraction improved to normal levels during convalescence.

Chopra in 2018 found reduced ejection fraction in 28.5% of their patients but this reduction in ejection fraction was transient and after 3 weeks of treatment, ejection fraction reverted to normal in all the cases.

Cardiac Manifestations in Dengue

In the present study the most common cardiac manifestation was bradycardia.There were 18 patients of bundle branch block (6.41%), 135 cases of bradycardia (48.04%), 3 cases of ectopics (1.07%), one case of left ventricular hypertrophy (0.36%), one case of mitral regurgitation (0.36%), 88 cases of myocardial injury (31.32%) and 2 patients of tachycardia (0.71%). 2 patients died during the course of their hospital stay, both these patients had myocardial injury and markedly reduced ejection fractions. One had an EF of 25% and the other had an EF of 20% with global hypokinesia.

Myocardial injury or myocarditis was reported in 35.5% cases by Chopra in 2018. Follow up echo revealed normal findings in all cases and the authors labelled myocardial involvement in dengue as acute reversible myocardial insult.

Satarasinghe et al in 2007 reported myocarditis in 24% cases of dengue fever. All their cases showed resolution of myocarditis on follow up echo examination indicating the transient and self-limiting nature of myocardial involvement.

OUTCOME OF CARDIAC MANIFESTATIONS

In the present study 2 patients with low ejection fractions of 20% and 25% succumbed to the disease. Rest of the patients were re-evaluated at the time of discharge and after 14 days of discharge by ECG and echocardiography. At the time of discharge bundle branch block was seen in 5 patients, one patient had left ventricular hypertrophy and one patient had mitral regurgitation. After 14 days, the bundle branch block cases also resolved spontaneously, but left ventricular hypertrophy and mitral regurgitation persisted. These statistically significant results showed that most of the cardiac manifestations resolved 14 days after discharge.

The fact that cardiac involvement in dengue is reversible is confirmed by the studies of Satarasinghe (2007), Chopra (2008), Tarique (2013) and Khongphatthanayothin (2003).

SUMMARY & CONCLUSION

- The present study was conducted in DMCH Ludhiana on 281 cases of dengue fever.
- This cross-sectional study aimed to study the spectrum of cardiac manifestations in dengue fever and the impact of these manifestations on patient outcome.
- Routine investigations of all patients like Hb, TLC, DLC, Platelet count, S.Creatinine, Bilirubin, SGOT, SGPT and chest X-ray were recorded.
- The diagnosis of cardiac manifestation was based principally on ECG, Echocardiography and cardiac biomarkers like CK-MB, Troponin-T, Myoglobin and lactate dehydrogenase (LDH). All patients were re-assessed at the time of discharge and 14 days post discharge to evaluate outcome of the cardiac manifestations. Myocardial injury was diagnosed by ST-T segment changes on ECG or regional wall motion abnormalities and/or reduced left ventricular ejection fraction on echocardiography or raised cardiac biomarkers.
- The age distribution of patients was from 10 to 69 years with the mean age of 41.9 years.
- There were 200 male patients in the study (71.17%) and 81 female patients (28.83%).
- 88.97% of patients showed thrombocytopenia. There was no significant co-relation between thrombocytopenia and cardiac manifestations.
- Similarly, no significant co-relation was found between leucopenia and cardiac manifestations.
- As regards co-morbid conditions, cardiac manifestations were more common in patients of chronic kidney disease, type II diabetes and hypertension. This co-relation was statistically significant with a p value of <0.001.

- The spectrum of cardiac manifestations in dengue included bundle branch block, bradycardia, ectopics, Myocardial injury, tachycardia, left ventricular hypertrophy and mitral regurgitation. Bradycardia was the most common cardiac manifestation.
- Two patients expired during the course of their hospital stay. Both had markedly reduced ejection fraction (20%& 25%).
- Most of the cardiac manifestations were reversible and rhythm abnormalities, ectopics and bundle branch block were transient and self-limiting in nature. The myocardial injury was also found to be reversible in majority of the patients. The results were statistically highly significant with a p value of <0.001.

CONCLUSIONS

The results of the present study indicate that myocardial involvement in dengue invariably runs a benign course and myocardial injury is reversible. The main tools to detect cardiac manifestations are ECG, Echocardiography and cardiac biomarkers. The spectrum of cardiac manifestations in this study included bradycardia, bundle branch blocks, ectopics, tachycardia, myocardial injury, left ventricular hypertrophy and mitral regurgitation. Most of these were reversible and did not have an adverse effect on patient outcome. A follow up study after 14 days is imperative to detect any residual disease or sequelae.

BIBLIOGRAPHY

1. World Health Organization (WHO) Global Strategy for Dengue Prevention and Control, 2012 – 2020, Geneva: WHO Press; 2012.
2. Gubler DJ, Meltzer M. Impact of dengue/dengue hemorrhagic fever on the developing world. Adv.Virus Res. 1999;53:35–70.
3. Dengue Cases and Deaths in the Country since 2010, NVBDCP
4. Halstead SB. Pathogenesis of dengue: challenges to molecular biology. Science. 1988;239:476-481.
5. Guzman MG, Halstead SB, Artsob H, Buchy P, Farrar J, Gubler DJ et al. Dengue: a continuing global threat. Nat Rev Microbiol. 2010;8:S7-16.
6. Moolani Y, Bukhman G, Hotez PJ. Neglected tropical diseases as hidden causes of cardiovascular disease. PLoS Negl Trop Dis. 2012;6:e1499.
7. Shivanthan MC, Navinan MR, Constantine GR, Rajapakse S. Cardiac involvement in dengue infection. J Infect Dev Ctries 2015;9:338-346.
8. Arora M, Patil RS. Cardiac Manifestation in Dengue Fever. J Assoc Physicians India. 2016;64:40-4.
9. Kularatne SA, GawarammanaI B, Kumarasiri PR. Epidemiology, clinical features, laboratory investigations and early diagnosis of dengue fever in adults: a descriptive study in Sri Lanka. Southeast Asian J of Trop Med & Pub Health. 2005;36:686-92.
10. Goroshi M, Chand D. Myocardial Performance Index (Tei Index): A simple tool to identify cardiac dysfunction in patients with diabetes mellitus. Indian Heart J. 2016;68:83-7.
11. Nimmagadda SS, Mahabala C, Boloor A, Raghuram PM, Nayak UA. Journal of Clinical and Diagnostic Research. Atypical Manifestations of Dengue Fever (DF) – Where Do We Stand Today? 2014;8:71-73.
12. Lee IK, Lee WH, Liu JW, Yang KD. Acute myocarditis in dengue hemorrhagic fever: a case report and review of cardiac

complications in dengue-affected patients. Int J. Infect Dis.2010;14:e919-22.

13. National Guidelines for Clinical Management of Dengue Fever 2014, NVBDCP.
14. Wali JP, Biswas A, Chandra S, Malhotra A, Aggarwal P, Handa R et al. Cardiac involvement in Dengue Haemorrhagic Fever. Int J Cardiol. 1998;64:31-6.
15. Kabra SK, Juneja R, Madhulika, Jain Y, Singhal T, Dar L, et al. Myocardial dysfunction in children with dengue haemorrhagic fever. Natl Med J India. 1998;11:59-61
16. Agarwal R, Kapoor S, Nagar R, Misra A, Tandon R, Mathur A et al. A clinical study of the patients with dengue hemorrhagic fever during the epidemic of 1996 at Lucknow, India. Southeast Asian J Trop Med Public Health. 1999;30:735-40.
17. Khongphatthanayothin A, Suesaowalak M, Muangmingsook S, Bhattarakosol P, Pancharoen C. Hemodynamic profiles of patients with dengue hemorrhagic fever during toxic stage: an echocardiographic study. Intensive Care Med. 2003;29:570-4.
18. Promphan W, Sopontammarak S, Pruekprasert P, Kajornwattanakul W, Kongpattanayothin A. Dengue myocarditis. Southeast Asian J Trop Med Public Health 2004; 35:611-3.
19. Singhi S, Kissoon N, Bansal A. Dengue and dengue hemorrhagicfever: Management issues in an intensive care unit. J Pediatr (Rio J). 2007;83:S22-35.
20. SatarasingheRL, Arultnithy K, Amarasena NL, BulugaHapitiya U, Sahayam DV. Asymptomatic myocardial involvement in a acute dengue virus infection in a cohort of adult Sri Lankans admitted to a tertiary referral centre. Br J Cardiol. 2007;14:171-3
21. Lateef A, Fisher D, Tambyah P. Dengue and Relative Bradycardia. Emerging Infectious Diseases 2007;13:651.
22. Kularatne SAM, Pathirage MMK, Kumarasiri PVR, Gunasena S, Mahindawanse SI. Cardiac complications of a dengue fever

outbreak in Sri Lanka, 2005. Trans R Soc Trop Med Hyg. 2007;101:804–8.

23. Wang CC, Wu CC, Liu JW, Lin AS, Liu SF, Chung YH, et al. Chest Radiographic Presentation in Patients with Dengue Hemorrhagic Fever Am. J. Trop. Med. Hyg. 2007;77:291–6.
24. Mahmod M, Darul ND, Mokhtar I, Nor NM, Anshar FM, Maskon O. Atrial fibrillation as a complication of dengue hemorrhagic fever: non-self-limiting manifestation. Int J Infect Dis. 2009;13:e316–18.
25. KaushikJS, Gupta P, Rajpal S, Bhatt S. Spontaneous resolution ofsinoatrial exit block and atrioventricular dissociation in a child with dengue fever. Singapore Med J 2010;51:e146‑8.
26. Salgado DM, Eltit JM, Mansfield K, Panqueba C, Castro D, Vega MR, et al. Heart and skeletal muscle are targets of dengue virus infection. Pediatr Infect Dis J 2010;29:238-42.
27. Gupta VK, Gadpayle AK. Subclinical cardiac involvement in dengue haemorrhagic fever. J Indian AcadClin Med.2010;11:107-11.
28. La-Orkhun V, Supachokchaiwattana P, Lertsapcharoen P, Khongphatthanayothin A. Spectrum of cardiac rhythm abnormalities and heart rate variability during the convalescent stage of dengue virus infection: a Holter study. Ann Trop Paediatr.2011;31:123-8.
29. Weerakoon KG, Kularatne SA, Edussuriya DH, Kodikara SK, Gunatilake LP, Pinto VG, et al. Histopathological diagnosis ofmyocarditis in a dengue outbreak in Sri Lanka, 2009. BMC Res Notes. 2011;4:268.
30. Azin FRFG, Gonçalves RP, Pitombeira MHS, Lima DM, Branco IC. Dengue: Profile of hematological and biochemical dynamics. Rev Bras Hemato lHemoter. 2012;34:36-41.
31. Yacoub S, Griffiths A, Chau T, Simmons C, Wills B, Hien T, et al. Cardiac function in Vietnamese patients with different dengue severity grades. Crit. Care Med. 2012;40:477–483.
32. Miranda CH, Borges MC, Matsuno AK, Vilar FC, Gali LG, Volpe GJ, *et al.* Evaluation of cardiac involvement during dengue viral infection. Clin. Infect. Dis. 2013;57:812–9.

33. Tarique S, Murtaza G, Asif S, QureshiIH. ECG manifestations in dengueinfection. Ann King Edward Med Univ (Print) 2013; 19:282-5.
34. Sam SS, Omar SF, Teoh BT, Abd Jamil J, Abu Bakar S. Review of dengue hemorrhagic fever fatal cases seen among adults: A retrospective study. PLoS Negl Trop Dis 2013;7:e2194.
35. SenguptaSP, Nugurwar A, Jaju R, Khandheria BK. Left ventricularmyocardial performance in patients with dengue hemorrhagic feverand thrombocytopenia as assessed by two-dimensional speckle tracking echocardiography. Indian Heart J 2013;65:276-82.
36. Marques N, Gan VC, Leo YS. Dengue myocarditis in Singapore: Twocase reports. Infection 2013;41:709-14.
37. Mallhi TH, Khan AH, Adnan AS, Sarriff A, Khan YH, Jummaat F. Clinico-laboratory spectrum of dengue viral infection and risk factors associated with dengue hemorrhagic fever: a retrospective study. BMC Infect Dis. 2015;15:399.
38. Shivanthan MC, Navinan MR, Constantine GR, Rajapakse S. Cardiac involvement in dengue infection. J Infect Dev Ctries 2015;9:338-346.
39. Kirawittaya T, Yoon IK, Wichit S, Green S, Ennis FA, Gibbons RV, et al. Evaluation of cardiac involvement in children with dengue by serialechocardiographic studies. PLoS Negl Trop Dis 2015;9:e0003943.
40. Lin SC, Huang CH, Lin CY, Lee WH, Su HM, Lin TH, et al. Dengue virus infection complicated with simultaneous multi-vessel ST elevation myocardial infarction. Journal of Microbiology, Immunology and Infection. 2016;49:619-620.
41. Dhanoa A, Hassan SS, Ngim CF, Lau CF, Chan TS, Adnan NAA. et al. Impact of dengue virus (DENV) co-infection on clinical manifestations, disease severity and laboratory parameters. BMC Infect Dis. 2016;16:406.

42. Wei HY, ShuPY and Hung MN. Characteristics and Risk Factors for Fatality in Patients with Dengue Hemorrhagic Fever, Taiwan, 2014. Am. J. Trop. Med. Hyg. 2016;95:322–7.
43. Virk HU, Inayat F, Rahman ZU. Complete heart block in association with dengue hemorrhagic fever. Korean Circ J. 2016;46:866-9.
44. Yadav RK, Kumar S. To study cardiac manifestations in patients presenting with dengue infection and to find out the correlation ofcardiac manifestations to warning signs of dengue. Int J Adv Med2017;4:323-8.
45. Yadav BL, Harshvardhan L, Yadav KS. Magnitude of cardiac involvement in dengue fever. J Evol Med Dent Sci 2017;6:570-4.
46. Kularatne SAM, Ralapanawa U, Dalugama C, Jayasinghe J, Rupasinghe S, Kumarihamy P. Series of 10 dengue fever cases withunusual presentations and complications in Sri Lanka: a single centre experience in 2016. BMC Infectious Diseases. 2018;18:674-681.
47. Papalkar PV, Sarode RR, Acharya S, Kumar S. Cardiac manifestations in dengue. Indian J Med Spec. 2019;10:30-4.
48. Kalakutakar A, Suresh H and Ashok G. Study of Cardiac Manifestations in Dengue Fever. Int.J.Curr.Microbiol.App.Sci. 2019;8:636-644.
49. Datta G, Mitra P. A study on cardiac Manifestations of Dengue fever. JAPI. 2019;67:14-16.
50. Lee IK, Hsieh CJ, Lee CT, Liu JW. Diabetic patients suffering dengue are at risk of developing Dengue shock syndrome/severe dengue: Emphasizing the impacts of co-existing co-morbidity(ies) and glycemic control on dengue severity. J MicrobiolImmunol Infect. 2020;53:69-78.

KEY TO MASTER CHART

1. Hb - Hemoglobin
2. TLC – Total Leucocyte count
3. DLC – Differential Leucocyte count
4. DLC (N) - Neutrophils
5. DLC (L) – Lymphocytes
6. DLC (E) – Eosinophils
7. DLC (B) – Basophils
8. PLT – Platelets
9. CR- Creatinine
10. CRCL – Creatinine clearance
11. Bilirubin T- Total
12. Bilirubin D – Direct
13. OT – SGOT
14. PT – SGPT
15. CXR – Chest X-Ray
16. HR – Heart Rate
17. BBB – Bundle branch block
18. RWMA – Regional wall motion abnormality
19. LVEF – Left ventricular ejection fraction
20. PAP – Pulmonary arterial pressure
21. CKMB- Creatinine kinase -MB
22. TRT- Troponin-T
23. Myo – Myoglobin
24. LDH – Lactate Dehydrogenase
25. ECG – Electrocardiogram
26. Echo – Echocardiography.

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
1	828064	S	32	M	2019071500	14.3	4000	64	27	6	3	120000	1.10	88.64	65	0.7	0.33	54	31	N	55	0.08	399	0.16	N	Imm in V2	tall T in V2	0.08	0	1	0	0
2	828065	A	40	F	20190715	13.6	4600	53	23	20	4	180000	0.70	101.19	60	0.6	0.27	637	318	R IN RLZ	58	0.08	447	0.12	N	nil	T I in v1-V3	0.04	0	1	0	0
3	828139	D	28	M	2019071536	14	4500	64	32	2	2	190000	0.60	191.85	74	0.6	0.2	45	30	N	86	0.08	418	0.12	N	nil	N	0.06	0	0	0	0
4	828157	K	22	M	2019071554	13	5000	66	31	2	1	150000	0.50	255.67	78	0.5	0.2	40	30	N	52	0.12	431	0.2	RBBB	2mm	Ti in V1, Tall T IN V2-V6	0.12	0	1	0	0
5	828120	R	19	M	2019071535	14.5	5500	66	31	2	1	200000	0.60	224.07	80	0.4	0.2	35	30	N	53	0.08	418	0.08	N	nil	N	0.08	0	1	0	0
6	828164	ID	46	F	2019071557	11	6600	64	30	4	2	250000	0.50	166.46	75	0.5	0.3	36	35	N	40	0.08	120	0.2	N	nil	N	0.12	0	2	0	0
7	766277	R	18	M	2019071567	14	5000	66	30	2	2	200000	0.20	610.00	72	0.2	0.1	32	34	N	93	0.06	437	0.12	NON SP	nil	N	0.08	0	0	0	0
8	828180	S	30	M	2019071577	12	4500	60	35	3	2	300000	0.10	1222.22	80	0.1	0.1	34	36	N	46	0.08	426	0.12	N	3mm	u wave in v1-v3	0.06	0	1	0	0
9	828168	B	60	M	2019071566	11	4800	62	34	2	2	250000	0.10	888.89	80	0.1	0.1	32	33	N	153	0.08	481	P absent	N	nil	N	0.08	0	0	1	0
10	828077	V	53	M	2019071510	16.4	4100	39	50	10	1	300000	0.20	453.13	75	0.3	0.1	63	42	N	42	0.12	433	0.12	RBBB	nil	N	0.08	0	1	0	0
11	827269	S	34	F	2019071156	11.1	5100	48	44	5	3	530000	0.60	156.42	75	0.6	0.3	224	177	N	48	0.08	437	0.12	N	nil	N	Ti in v1-v4 isoelectric T in v5,v6	0	1	0	0
12	825693	S	18	M	2019070559	14.5	5300	50	39	5	6	600000	0.70	183.97	76	3	2.1	159	50	N	79	0.08	419	0.12	N	nil	Ti in v1-v4	0.04	0	0	43w2q	0
13	827801	S	68	F	2019071405	11.3	4500	49	39	10	2	250000	0.70	97.14	80	0.5	0.2	49	71	N	46	0.08	415	0.12	N	nil	Ti in v1 v2	0.08	0	1	0	0
14	827363	H	63	M	2019071236	14.9	7900	77	11	10	2	400000	1.10	53.47	55	1	0.4	60	63	PE Rt	45	0.08	395	0.12	N	nil	N	0.04	0	1	0	0
15	821778	P	75	M	2019071367	8	10800	47	39	8	6	300000	0.90	75.23	75	0.7	0.3	153	54	N	78	0.08	390	0.12	N	nil	N	0.04	0	0	0	0
16	828051	A	54	M	2019071480	12.9	5800	84	10	4	2	280000	0.80	122.43	82	0.5	0.2	171	70	N	46	0.08	419	0.12	N	nil	deep S, small T	0.04	0	1	0	0
17	827207	LD	58	F	2019071119	12.4	4300	51	38	9	2	300000	0.80	102.86	85	0.5	0.1	445	179	PE B/L	104	0.08	440	0.12	N	nil	N	0.12	0	0	0	0
18	827201	P	36	M	2019071116	13.2	10900	50	40	8	2	170000	1.20	95.09	79	0.8	0.1	295	190	N	44	0.8	431	0.12	N	nil	N	0.04	0	1	q3, t3 in lead III	0
19	825325	R	43	M	2019070329	13.8	13800	76	14	3	7	68000	0.90	116.76	78	0.6	0.1	119	50	N	45	0.08	397	0.12	N	nil	N	0.08	0	1	0	0
20	827329	R	44	M	2019071208	15	5090	50	39	8	3	15000	1.00	114.67	86	0.4	0.1	316	220	N	105	0.08	436	0.12	N	nil	N	0.12	0	0	0	0
21	827369	M	60	M	2019071239	13.5	4000	53	34	10	3	40000	0.80	106.94	77	0.4	0.2	174	70	N	48	0.12	407	0.12	NON SP	1mm in vi,v2	N	0.08	0	1	0	0
22	825698	R	32	M	2019070560	16	3700	55	33	10	2	19000	1.00	112.50	75	0.7	0.3	252	184	N	44	0.08	389	0.12	N	nil	N	0.08	0	1	0	0
23	825415	D	66	M	2019070435	14.8	3400	59	29	10	2	32000	0.80	110.49	86	1.3	0.8	235	129	PE Rt	77	0.08	417	0.12	N	nil	N	0.12	0	0	0	0
24	827342	P	28	M	2019071214	13.1	6800	44	43	9	4	50000	1.00	129.11	83	0.5	0.2	74	17	N	45	0.08	441	0.12	N	nil	N	0.08	0	1	0	0
25	825173	R	54	M	2019070278	14.9	8100	44	47	6	3	21000	1.20	77.64	78	0.2	0.1	118	102	N	46	0.08	405	0.12	N	nil	N	0.04	0	1	0	0
26	828141	S	72	M	2019071540	12.6	5000	63	25	10	2	18000	1.10	72.98	85	0.3	0.1	106	86	R B/L	94	0.08	433	0.12	N	nil	N	0.08	0	0	0	0
27	825726	A	25	M	2019070573	14.5	3200	46	42	10	2	54000	0.90	127.78	72	0.2	0.07	57	41	N	44	0.08	373	0.08	N	nil	N	0.08	0	1	0	0
28	826256	P	38	M	2019070764	16.2	5800	48	40	10	2	60000	0.80	138.13	78	0.6	0.3	304	179	N	42	0.04	410	0.16	N	nil	N	0.04	0	1	0	0
29	827932	J	35	M	2019071417	14.1	1500	50	40	8	2	17000	0.70	137.50	66	0.5	0.1	287	229	N	65	120	360	0.12	N	nil	Ti in v1-v3	0.08	0	0	0	0
30	826505	S	31	F	2019070877	10.7	5100	50	40	7	3	74000	0.50	164.71	64	0.6	0.2	284	167	N	44	0.08	423	0.12	N	nil	Ti in v1-v2	0.04	0	1	0	0
31	108085	M	48	M	2019071247	15.6	8800	45	39	12	4	17000	0.80	121.39	76	0.5	0.2	98	38	N	42	0.08	463	0.08	RBBB	nil	Ti in v1-v6	0.02	0	1	0	0
32	824488	DP	20	M	2019069980	18.6	1600	62	25	11	2	8000	1.20	104.17	75	1.8	0.8	7159	3203	N	91	0.08	448	0.12	N	nil	N	0.08	0	0	0	0
33	825279	T	35	F	2019070312	11.3	3500	49	39	9	3	126000	0.60	134.29	65	0.5	0.15	211	89	N	44	0.2	417	0.08	n	nil	poor r wave progr	0.08	0	1	0	0
34	826461	B	37	M	2019070829	16.5	3600	70	23	5	2	60000	0.70	159.40	78	0.4	0.12	116	109	N	89	0.08	401	0.12	N	nil	N	0.08	0	0	0	0
35	827404	M	65	F	2019071267	8.1	14500	71	21	4	4	31000	3.01	16.18	55	0.4	0.08	72	19	B/L LZ Hazy	46	0.08	433	0.12	N	nil	N	0.04	0	1	0	0
36	826455	D	32	M	2019070832	15.3	9500	69	19	10	2	36000	0.80	163.13	87	0.6	0.3	185	97	N	42	0.1	463	0.08	N	nil	N	0.08	0	1	0	0
37	827302	A	60	M	2019071182	14.7	6200	47	43	7	3	10000	1.20	60.19	65	0.9	0.3	179	97	N	63	0.08	443	0.12	N	nil	N	0.08	0	0	0	0
38	827729	V	32	M	2019071363	14.3	7000	64	27	8	1	24000	1.10	107.73	79	1.4	0.6	335	177	PE Lt	93	0.08	452	0.12	N	nil	N	0.04	0	0	0	0
39	826734	M	24	M	2019071022	13.7	2400	53	38	7	2	52000	0.90	152.16	85	0.3	0.1	42	41	N	41	120	408	0.12	non sp	nil	N	0.08	0	1	0	0
40	827380	P	68	F	2019071250	14	9600	51	37	10	2	20000	0.80	72.25	68	0.5	0.1	195	87	PE Lt	42	0.08	388	0.12	N	nil	N	0.04	0	1	0	0
41	761789	J	56	M	2019071130	12.3	2500	59	29	10	2	97000	1.10	80.61	76	0.2	0.09	162	122	N	76	0.04	394	0.12	N	nil	N	0.04	0	0	0	0
42	827209	P	63	M	20171111	14	1900	44	44	8	4	20000	0.60	114.07	64	0.5	0.1	151	66	PE Rt	43	0.08	420	0.12	N	nil	N	0.04	0	1	0	0
43	833258	D	28	M	2019073911	15	4000	34	53	10	3	58000	0.55	220.61	78	0.2	0.1	229	104	N	89	0.08	430	0.14	non sp	nil	n	0.04	0	0	0	0
44	834092	H	35	F	2019074269	12	4000	51	35	10	4	52000	1.00	80.57	65	0.4	0.2	245	98	N	42	0.04	406	0.12	N	nil	N	0.12	0	1	0	0
45	783751	J	48	M	2019074599	12	1600	62	28	10	0	52000	0.80	122.99	77	1	0.4	88	54	N	44	120	461	0.12	N	nil	N	0.04	0	1	0	0
46	831505	A	70	F	2019073116	12	5700	60	24	10	6	112000	1.10	41.32	55	0.9	0.2	68	44	N	68	0.08	412	0.12	non sp	nil	N	0.04	0	0	0	0
47	834431	S	30	M	2019074508	14	6900	70	20	10	0	26000	0.50	250.56	82	0.6	0.1	113	40	N	46	0.08	393	0.12	non sp	nil	N	0.08	0	1	0	tall T in v1-v6
48	833013	R	20	M	2019073746	15.4	2600	38	49	10	3	80000	0.70	171.43	72	0.5	0.1	27	16	N	42	0.08	394	0.12	N	nil	N	0.08	0	1	0	0
49	833015	S	52	F	2019074210	13.9	9700	50	40	10	0	52000	0.70	112.79	76	0.7	0.1	181	118	N	64	0.08	433	0.12	N	nil	N	120	0	0	0	0
50	835028	T	35	M	2019074700	15.9	5300	45	40	10	5	42000	0.60	187.15	77	0.8	0.2	30	35	N	44	0.08	413	0.08	N	nil	N	0.08	0	1	0	0
51	528100	M	37	F	2019073708	11	6300	53	34	10	3	130000	0.60	162.13	80	0.2	0.1	208	294	N	42	0.08	453	0.12	N	nil	N	0.08	0	1	0	0
52	832999	A	18	M	2019073709	14.2	7800	40	46	10	4	51000	0.37	311.41	68	0.5	0.2	479	334	N	45	0.04	391	0.16	non sp	nil	N	0.08	0	1	0	0
53	833150	P	46	M	2019073810	17.3	2900	52	37	7	4	25000	1.04	102.94	82	0.3	0.1	75	37	N	43	0.08	430	0.12	N	nil	N	0.04	0	1	0	0

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge													14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhthymias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
1	828064	N	N	N	p	N	60	0	N	5	0	100	25	140	myo	92	0.08s	399	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
2	828065	N	N	N	p	N	60	0	N	5	0	80	25	140	myo	81	0.08s	447	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
3	828139	N	N	N	P	N	60	0	N	6	0	150	25	180	normal	86	0.08S	418	0.12	N	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	Nil
4	828157	N	N	N	N	N	60	0	N	5	0	150	40	140	myo RBBB, brady	66	0.12	431	0.2	RBBB	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	DM2
5	828120	N	N	N	N	N	60	LVH	N	5	0	200	25	140	brady	81	0.08S	418	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
6	828164	N	N	N	P	N	60	0	N	6	0	200	25	230	brady	62	0.08s	120	0.2	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	DM2
7	766277	N	N	N	P	N	60	0	N	6	0	200	50	140	myo	93	0.06s	437	0.12	NON SP	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
8	828180	N	N	N	P	N	60	0	N	5	0	200	25	140	myo, brady	95	0.08s	426	0.12	N	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	HTN
9	828168	N	N	N	P	N	60	0	N	6	0	200	60	240	tachy	96	0.08	481	P absent	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
10	828077	N	N	N	P	N	60	0	N	7	0	200	25	140	RBBB, brady	64	0.12	433	0.12	RBBB	nil	N	0.08	0	0	0	BBB	N	N	NIL	Normal	Nil
11	827269	N	N	mild MR	P	N	62	0	N	20.3	0.04	505	29	300	myo, brady	78	0.08	437	0.02	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
12	825693	N	N	N	P	N	60	0	N	5	0	150	28	150	myo	79	0.08	419	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
13	827801	N	N	N	P	N	60	0	N	5	0	150	28	120	myo, brady	71	0.08	415	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
14	827363	N	N	N	P	N	60	0	N	7	0	150	30	150	brady	95	0.08	395	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
15	821778	N	N	N	P	N	61	0	N	8	0	150	28	140	normal	76	0.08	395	0.12	N	nil	N	0.09	0	0	0	NORMAL	N	N	NIL	Normal	DM2
16	828051	N	N	N	P	N	60	0	N	6	0	150	28	150	brady	63	0.08	419	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
17	827207	N	N	N	P	N	60	0	N	9	0	150	40	150	normal	95	0.08	440	0.1	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	CLD
18	827201	N	N	N	P	N	60	0	N	6	0	150	28	130	brady	98	0.8	431	0.12	N	nil	N	0.14	0	0	0	NORMAL	N	N	NIL	Normal	DM2
19	825325	N	N	N	P	N	60	0	N	7	0	150	30	150	brady	73	0.08	397	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
20	827329	N	N	N	P	N	62	0	N	6	0	150	28	150	normal	100	0.08	436	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
21	827369	N	N	N	P	N	60	0	N	6	0	150	28	170	myo, brady	63	0.12	407	0.12	NON SP	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
22	825698	N	N	N	P	N	60	0	N	6	0	150	35	150	brady	68	0.08	389	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CLD
23	825415	N	N	N	P	N	65	0	N	5	0	180	28	150	normal	77	0.08	417	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
24	827342	N	N	N	P	N	60	0	N	5	0	150	28	160	brady	77	0.08	441	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
25	825173	N	N	N	P	N	58	0	N	5	0	160	40	150	brady	71	0.08	405	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
26	828141	N	N	N	P	N	62	0	N	5	0.5	1820	348	400	normal	94	0.08	433	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
27	825726	N	N	N	P	N	62	0	N	5	0	200	30	120	brady	61	0.08	373	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
28	826256	N	N	N	P	N	62	0	N	5	0	200	35	140	brady	96	0.04	410	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
29	827932	N	N	N	P	N	60	0	N	25.6	0.5	104	225	130	myo	61	120	360	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
30	826505	N	N	N	P	N	62	0	N	16	0.5	100	40	130	myo, brady	100	0.08	423	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
31	108085	N	N	N	P	N	62	0	N	5	0.5	1940	168	130	myo, RBBB, brady	74	0.08	463	0.08	RBBB	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
32	824488	N	N	N	P	N	64	0	N	8	0	100	30	140	normal	91	0.08	448	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CLD
33	825279	N	N	N	P	N	62	0	N	6	0	150	35	130	myo, brady	90	0.2	417	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
34	826461	N	N	N	P	N	62	0	N	6	0	160	28	180	normal	89	0.08	401	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
35	827404	N	N	N	P	N	55	0	N	5	0	100	29	160	brady	82	0.08	433	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
36	826455	N	N	N	P	N	62	0	N	3409	0.5	1110	500	400	brady	95	0.1	463	0.18	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
37	827302	N	N	N	P	N	62	0	N	6	0	180	28	190	normal	64	0.08	443	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
38	827729	N	N	N	P	N	62	0	N	6	0	180	28	190	normal	93	0.08	452	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
39	826734	N	N	N	P	N	60	0	N	5	0	180	40	180	BBB, brady	97	120	408	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
40	827380	N	N	N	P	N	62	0	N	6	0	180	28	210	brady	65	0.08	388	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
41	761789	N	N	N	N	N	58	0	N	5	0	180	36	190	normal	76	0.04	394	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
42	827209	N	N	N	P	N	62	0	N	5	0	180	28	190	brady	75	0.08	420	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
43	833258	N	N	N	P	N	58	0	N	7	0	180	40	200	BBB	89	0.08	430	0.14	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
44	834092	N	N	N	P	N	62	0	N	7	0	180	28	190	brady	83	0.04	406	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	DM2
45	783751	N	N	N	P	N	62	0	N	6	0	180	30	190	brady	100	120	461	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
46	831505	N	N	N	P	N	62	0	N	5	0	180	28	190	BBB	68	0.08	412	0.12	N	nil	N	0.04	0	0	0	NORMAL	BBB	N	NIL	Normal	Nil
47	834431	N	N	N	P	N	62	0	N	5	0	180	28	230	myo, BBB, brady	72	0.08	393	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
48	833013	N	N	N	P	N	62	0	N	6	0	180	40	190	brady	76	0.08	396	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
49	833015	N	N	N	P	N	62	0	N	6	0	170	28	210	normal	64	0.08	433	120	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
50	835028	N	N	N	P	N	62	0	N	5	0	180	40	190	brady	100	0.08	413	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
51	528100	N	N	N	P	N	62	0	N	5	0	160	28	220	brady	84	0.08	453	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
52	832999	N	N	N	P	N	60	0	N	8	0	150	30	190	BBB, brady	96	0.04	391	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
53	833150	N	N	N	P	N	62	0	N	8	0	200	28	190	brady	78	0.08	430	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
54	835346	A	42	M	2019079448	15.2	8500	56	28	10	6	110000	1.10	98.99	80	1.4	0.7	104	114	N	84	0.12	437	0.12	non sp	nil	N	0.1	0	0	0	0
55	833048	R	43	M	2019073742	14.5	2100	53	34	10	3	158000	0.70	150.12	78	0.2	0.01	68	101	N	75	0.08	430	0.08	N	nil	N	0.08	0	0	0	0
56	38044	K	20	F	2019073900	9	4900	63	27	8	2	90000	0.70	125.48	62	0.3	0.1	53	42	N	42	0.08	454	0.12	N	nil	N	0.04	0	1	0	0
57	832457	P	63	F	2019073542	11	6100	69	17	10	4	70000	0.70	98.69	76	0.6	0.5	353	191	PE Rt	41	0.08	575	0.12	N	nil	N	0.04	0	1	0	0
58	832401	S	52	F	2019073489	10.6	9400	34	53	10	3	30000	0.40	223.36	86	1.1	0.4	362	166	N	42	0.04	422	0.12	N	nil	N	0.04	0	1	0	0
59	832399	K	53	F	2019073483	13.9	10800	60	30	8	2	15000	1.05	82.17	84	0.4	0.15	1045	375	N	46	0.08	436	0.12	N	nil	N	0.04	0	1	0	0
60	832216	A	27	M	2019073346	11.3	5100	80	10	8	2	41000	1.00	119.28	76	2.1	1.8	482	543	PE b/l b/l infil	44	0.08	426	0.12	N	nil	N	0.04	0	1	0	0
61	834744	J	38	M	2019074521	11.5	3900	63	21	15	1	48000	3.20	34.53	78	5.1	2.4	6304	2605	PE b/l b/l infil	136	0.08	451	0.08	N	nil	isoelectric ST	0.04	0	0	0	0
62	833139	K	40	F	2019073806	9.8	16400	65	28	6	1	120000	1.40	60.71	72	5.5	2.9	3049	1541	PE b/l b/l infil	43	0.08	466	0.12	N	nil	N	0.04	0	1	0	0
63	833140	K	55	M	2019073997	12.1	7000	70	20	8	2	48000	0.90	89.20	68	11.4	6.8	673	571	PE b/l b/l infil	46	0.04	431	0.12	N	nil	N	0.04	0	1	0	0
64	834930	S	18	M	2019074651	14	8900	44	45	10	1	22000	0.67	187.15	74	1.04	0.23	140	134	N	83	0.04	440	0.12	N	nil	N	0.08	0	0	0	0
65	834028	B	19	M	2019074224	13	4600	51	39	8	2	60000	0.20	655.42	78	0.59	0.3	31	24	PE b/l b/l infil	42	0.08	402	0.12	N	nil	N	0.04	0	1	0	0
66	82992	P	42	M	2019073605	12	9800	77	13	8	2	50000	0.80	132.71	78	5.2	3.09	414	283	N	43	0.08	403	0.12	N	nil	N	0.04	0	1	0	0
67	833032	H	80	M	2019073731	7.7	17500	85	6	8	1	83000	5.10	10.62	65	0.5	0.4	585	371	b/l infil	44	0.08	439	0.12	N	nil	N	0.08	0	1	0	0
68	831712	R	55	M	2019073163	12	3000	71	19	6	4	40000	0.90	85.26	65	3.7	1.8	628	506	PE b/l	64	0.08	441	0.2	N	nil	N	0.04	0	0	0	0
69	828050	A	24	F	2019071477	9.3	14300	69	18	9	4	60000	0.80	99.28	58	1.2	0.5	37	110	N	44	0.08	412	0.08	N	nil	N	0.08	0	1	0	0
70	834114	S	30	M	2019074302	13.2	3000	53	37	10	0	53000	0.70	170.24	78	0.9	0.2	227	170	N	46	0.12	424	0.12	N	nil	N	0.04	0	1	0	0
71	834174	V	40	M	2019074340	16.9	10900	42	40	16	2	24000	0.10	1111.11	80	0.5	0.1	232	39	N	48	0.08	430	0.12	N	nil	N	0.04	0	1	0	0
72	833297	N	42	M	2019073957	16.3	1600	49	38	10	3	36000	1.10	94.04	76	0.4	0.1	263	208	N	82	0.12	422	0.12	N	nil	N	0.08	0	0	0	0
73	833304	D	30	M	2019073961	12.1	10600	58	28	10	4	65000	0.79	164.38	85	0.6	0.5	128	32	PE b/l	99	0.08	449	0.12	N	nil	N	0.08	0	0	0	0
74	833236	V	75	F	2019073901	9.3	6700	75	11	10	4	63000	0.80	62.35	65	0.6	0.2	1180	626	PE b/l nod b/l	44	0.04	445	0.12	N	nil	Ti in v1-v6	Ti in v1-v6	0	1	0	0
75	852517	PL	80	M	2019082831	12.9	5100	40	45	12	3	50000	0.50	113.33	68	0.5	0.1	81	35	Ret b/l	46	0.12	453	0.12	N	nil	Tall T in v2-v5	0.08	0	1	0	0
76	849472	S	47	F	2019081384	10.9	8300	76	18	3	3	90000	0.70	112.93	72	0.5	0.1	133	55	Ret b/l	88	0.08	413	0.12	N	nil	N	0.08	0	0	0	0
77	848710	B	80	F	2019081045	10	9800	60	25	12	3	43000	4.59	8.64	56	0.8	0.4	185	77	b/l nod	46	0.04	448	0.12	N	nil	N	0.04	0	1	0	0
78	848781	V	65	M	2019081094	10	4100	75	14	10	1	92000	0.80	101.56	78	3.2	1.7	97	77	PE b/l nod b/l	43	0.08	464	0.12	N	nil	isoelectric T wave	isolectric T wave	0	1	0	Qs in v4-v6 poor R progression biphasic P in V1
79	847606	ML	32	M	2019080530	13.4	8500	55	36	6	3	15000	0.10	1215.00	81	0.6	0.2	203	72	N	67	0.08	392	0.12	N	nil	ST i in v2-v6 Ti in v2-v6	Ti in v2-v6	0	0	0	Q in lead III
80	849466	S	43	M	2019081383	11.6	6400	58	30	10	2	110000	0.60	188.61	84	2.2	1.8	265	127	b/l nod	44	0.12	449	0.12	non sp	nil	Ti in v1-v3	0.08	0	1	0	0
81	847577	R	55	F	201908499	11.8	7600	66	23	8	3	30000	0.70	106.08	74	1.2	0.8	427	321	PE Lt	44	0.12	420	0.12	N	nil	Ti in all	0.04	0	1	0	0
82	846706	V	18	M	2019080130	14.1	9500	69	20	10	1	14000	0.90	135.56	72	1.2	0.7	1180	453	b/l PE	106	0.04	404	0.08	N	nil	N	0.04	0	0	0	0
83	845822	H	55	M	2019079697	9.5	3600	62	25	10	3	40000	0.60	167.25	85	2.5	1.9	74	39	PE b/l b/l infil	42	0.04	482	0.16	N	nil	N	0.08	0	1	0	0
84	849470	H	45	M	2019081386	11	4500	63	26	10	1	60000	1.20	83.56	76	2.2	1.2	80	65	N	70	0.04	418	0.12	N	nil	N	0.08	0	0	0	0
85	849471	S	56	F	2019081382	12	5600	70	28	1	1	62000	1.30	64.84	85	2.1	1.3	70	68	N	43	0.04	433	0.12	N	nil	N	0.06	0	1	0	0
86	848795	H	38	F	2019081105	12.6	9700	49	43	4	4	30000	0.90	88.31	66	0.7	0.2	386	231	N	44	0.08	455	0.12	N	nil	N	0.02	0	1	0	Isoelectric T in v1-v6
87	849430	SL	65	F	2019081358	10	4500	42	48	6	4	30000	0.80	70.83	64	0.5	0.1	50	35	N	42	0.08	442	0.12	N	nil	N	0.04	0	1	0	0
88	848900	ML	60	M	2019081154	16	7600	68	19	10	3	14000	1.10	68.69	68	0.5	0.3	31	30	PE Lt	98	0.08	442	0.12	n	nil	n	0.04	0	0	0	0
89	849476	SL	42	M	2019081387	12	4500	62	27	10	1	40000	1.30	79.57	76	0.6	0.4	342	187	N	43	0.08	381	0.12	N	nil	N	0.08	0	1	0	0
90	849453	R	25	F	2019081375	12	14100	60	36	2	2	28000	1.00	92.32	68	0.5	0.1	137	77	N	42	0.04	444	0.12	N	1mm in v2-v6	tall peaks in v2-v6	0.08	0	1	0	upright QRS IN V1-V6
91	794329	M	24	M	2019058190	8.5	12500	87	10	2	1	60000	1.50	83.78	78	0.7	0.6	152	170	PE b/l b/l infil	64	0.08	403	0.12	N	IN V2-V4	N	0.04	0	0	0	0
92	177978	S	54	F	2019063900	8.9	2000	92	4	3	1	90000	7.80	10.02	77	0.2	0.17	197	189	b/l PE	41	0.04	542	0.12	N	nil	isoelectric	0.08	0	1	0	0
93	817324	M	46	F	2019066984	10.4	18600	53	34	10	3	71000	0.60	138.72	75	1.3	1.1	1250	2191	PE b/l b/l infil	42	0.08	445	0.12	N	nil	N	0.04	0	1	0	0
94	818549	G	35	M	2019067478	16	5100	61	27	10	2	60000	1.00	99.17	68	0.2	0.1	176	242	N	76	0.08	405	0.12	N	nil	N	0.04	0	0	0	0
95	318880	D	36	M	2019067587	13.3	5300	57	30	10	3	60000	1.10	105.05	80	0.5	0.1	185	103	N	41	0.04	443	0.12	N	nil	N	0.04	0	1	0	0

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge												14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhythmias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
54	835346	N	N	N	P	N	62	0	N	8	0	180	40	200	BBB	84	0.12	437	0.12	N	nil	N	0.1	0	0	0	NORMAL	N	N	NIL	Normal	HTN
55	833048	N	N	N	P	N	62	0	N	5	0	189	28	190	normal	75	0.08	438	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
56	38044	N	N	N	P	N	58	0	N	5	0	190	32	180	brady	78	0.08	454	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
57	832457	N	N	N	P	N	62	0	N	6	0	190	28	190	brady	98	0.08	575	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
58	832401	N	N	N	P	N	62	0	N	6	0	180	50	270	brady	96	0.04	422	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
59	832399	N	N	N	P	N	62	0	N	7	0	180	28	190	brady	80	0.08	436	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
60	832216	N	N	N	P	N	62	0	N	5	0.5	3360	40	190	brady	96	0.08	426	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
61	834744	N	N	N	P	N	62	0	N	8.8	0.5	3670	30	170	normal	95	0.08	451	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
62	833139	N	N	N	P	N	62	0	N	5	0	180	40	190	brady	99	0.08	466	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
63	833140	N	N	N	P	N	60	0	N	6	0	180	35	190	brady	85	0.04	431	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
64	834930	N	N	N	P	N	62	0	N	5	0	170	26	200	normal	83	0.04	440	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
65	834028	N	N	N	P	N	60	0	N	5	0	100	28	190	brady	71	0.08	402	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
66	82992	N	N	N	P	N	62	0	N	6	0	180	25	190	brady	88	0.08	403	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
67	833032	N	N	N	P	N	62	0	N	5	0.05	1230	310	190	brady	82	0.08	439	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
68	831712	N	N	N	P	N	62	0	N	5	0	100	30	180	normal	64	0.08	441	0.2	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
69	828050	N	N	N	P	N	62	0	N	5	0	150	40	170	brady	83	0.08	412	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
70	834114	N	N	N	P	N	55	0	N	5	0	150	50	190	brady	80	0.12	424	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
71	834174	N	N	N	P	N	62	0	N	5	0	150	55	190	brady	86	0.08	430	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
72	833297	N	N	N	P	N	58	0	N	5	0	150	40	160	normal	82	0.12	422	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
73	833304	N	N	N	P	N	62	0	N	6	0	150	25	190	normal	99	0.08	449	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
74	833236	N	N	N	P	N	62	0	N	6	0	200	28	200	brady	100	0.04	445	0.12	N	nil	N	Ti in v1-v6	0	0	0	NORMAL	N	N	NIL	Normal	DM2
75	852517	N	N	N	P	N	62	0	N	5	0.05	532	99	190	brady	75	0.12	453	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
76	849472	N	N	N	P	N	62	0	N	5	0.01	430	110	190	normal	88	0.08	413	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
77	848710	N	N	N	P	N	62	0	N	5	0.05	2170	500	210	brady	74	0.04	448	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
78	848781	P	N	MR	P	P	25	0	N	5	0.05	1460	87	900	myo, MR, brady	-	-	-	-	-	-	-	-	-	-	-	mortality	-	-	Mortality	mortality	HTN
79	847606	N	N	N	P	N	50	0	N	30	0	800	100	180	myo	67	0.08	392	0.12	N	nil	N	Ti in v2-v6	0	0	0	NORMAL	N	N	NIL	Normal	HTN
80	849466	N	N	N	P	N	50	0	N	5	0.05	2400	124	600	myo, brady	85	0.12	449	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
81	847577	N	N	TR	P	N	62	0	N	5	0.02	1270	89	500	myo, brady	88	0.12	420	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
82	846706	N	N	N	P	N	50	0	N	5	0	200	26	200	normal	100	0.04	404	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
83	845822	N	N	N	P	N	60	0	N	5	0	180	27	200	brady	98	0.04	482	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
84	849470	N	N	N	P	N	50	0	N	5	0	200	28	150	normal	70	0.04	418	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
85	849471	N	N	N	P	N	65	0	N	5	0.05	2820	70	400	myo, brady	98	0.04	433	0.12	N	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	HTN
86	848795	N	N	N	P	N	60	0	N	5	0	170	30	150	brady	99	0.08	455	0.12	N	nil	N	0.02	0	0	0	NORMAL	N	N	NIL	Normal	DM2
87	849430	N	N	N	P	N	56	0	N	5	0.05	2560	87	500	brady	78	0.08	442	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
88	848900	N	N	N	P	N	50	0	N	5	0	180	40	450	normal	98	0.08	442	0.12	n	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
89	849476	N	N	N	P	N	50	0	N	6	0	190	40	350	myo, brady	62	0.08	381	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
90	849453	N	N	N	P	basal septum akinetic	60	0	N	7	0	200	30	280	myo, brady	84	0.04	444	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
91	794329	N	moderate	N	P	global	38	0	N	8	0	180	40	280	myo	66	0.08	403	0.12	N	nil	N	0.04	0	0	0	NORMAL	EF	N	NIL	Normal	HTN
92	177978	N	N	N	P	global	20	0	N	6	0	190	40	210	myo, brady	-	-	-	-	-	-	-	-	-	-	-	Mortality	-	-	mortality	mortality	DM2
93	817324	N	N	mild MR	P	N	60	0	N	5	0.05	269	74.8	200	MR, brady	96	0.08	445	0.12	N	nil	N	0.04	0	0	0	MR	N	N	NIL	MR	Nil
94	818549	N	N	N	P	N	60	0	N	5	0	180	50	140	normal	76	0.08	405	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
95	318880	N	N	N	P	N	60	0	N	5	0	200	55	150	brady	86	0.04	443	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
96	818907	G	88	F	2019067612	12	6800	60	30	8	2	78000	0.90	46.38	68	0.3	0.1	78	49	N	42	0.08	464	0.12	N	nil	N	0.08	0	1	0	0
97	818844	K	35	M	2019067565	11.6	5200	35	50	10	5	34000	0.60	184.72	76	0.6	0.1	273	259	N	79	0.08	419	0.12	N	nil	N	0.08	0	0	0	0
98	786763	J	26	M	2019067436	15.2	3300	64	24	10	2	196000	1.10	112.27	78	1.1	0.2	66	19	N	43	0.04	400	0.12	non sp	nil	N	0.08	0	1	0	0
99	818090	B	28	M	2019067246	14.6	5000	50	37	10	3	78000	1.10	107.47	76	1.1	0.1	122	144	N	44	0.04	339	0.2	N	nil	N	0.06	0	2	0	0
100	819490	M	17	M	2019067776	14	6000	55	35	10	0	55000	0.90	136.67	72	1	0.2	140	135	N	46	0.08	398	0.12	N	4mm in all precordial leads	4mm in all precordial leads	0.2	0	1	0	0
101	818759	S	26	F	2019067494	10	8900	60	25	12	3	30000	0.10	915.17	68	0.5	0.2	265	134	N	41	0.04	428	P absent	N	nil	N	P absent	0	1	0	0
102	816438	RK	56	M	2019066607	12	7600	68	20	10	2	60000	0.60	151.67	78	0.4	0.1	299	245	N	43	0.08	433	0.12	N	nil	N	0.12	0	1	0	0
103	816411	P	50	M	2019066592	13	6200	48	38	10	4	34000	0.70	157.14	88	0.4	0.1	121	50	N	41	0.08	418	0.12	N	nil	N	0.12	0	1	0	0
104	816404	A	40	M	2019066606	13	3200	55	30	10	5	46000	1.30	87.61	82	2.2	1.1	145	54	N	72	0.08	407	0.12	N	nil	N	0.08	0	0	0	0
105	817979	K	45	M	2019067186	11.8	5300	50	40	9	1	41000	0.70	162.10	86	0.7	0.2	180	95	N	40	0.08	417	0.12	N	nil	N	0.04	0	1	0	0
106	815484	M	67	F	2019066311	9.1	5500	45	30	25	0	53000	0.70	96.03	78	0.4	0.1	252	134	PE Lt	41	0.04	442	0.12	N	nil	N	0.04	0	1	0	0
107	818200	V	67	M	2019067320	13.1	8100	60	37	3	0	28000	0.60	109.84	65	1.4	0.6	222	153	N	42	0.08	400	0.12	N	nil	N	0.04	0	1	0	0
108	818170	SR	40	M	2019067298	13.9	12300	70	27	2	1	22000	0.80	135.42	78	0.4	0.2	299	190	N	61	0.12	382	0.12	non sp	2mm in v2 1mm in v3-v6	tall T in v2, v3	0.08	0	0	0	0
109	818015	J	46	M	2019067206	12.3	3300	55	33	10	2	60000	0.80	138.72	85	0.4	0.1	77	45	N	85	0.08	399	0.12	N	nil	N	0.04	0	0	0	0
110 neelam	818156	N	58	F	2019067287	10	5100	53	35	10	2	5000	0.70	107.87	78	0.3	0.11	38	30	b/l PE	43	0.08	449	0.16	N	nil	N	0.08	0	1	0	0
111 jagtar	817366	J	32	M	2019067008	12.9	3700	38	50	10	2	60000	0.78	146.15	76	0.9	0.6	333	185	Rt PE	42	0.12	448	0.12	non sp	nil	N	0.06	0	1	0	0
112	817484	S	38	M	2019067555	15.5	2200	65	23	10	2	20000	0.50	232.33	82	0.8	0.2	294	521	N	87	0.08	399	0.12	N	nil	N	0.08	0	0	0	0
113	817353	R	35	M	2019066999	13.8	5300	34	52	10	4	46000	0.70	156.25	75	0.7	0.3	202	72	N	43	0.12	400	0.2	RBBB	nil	N	0.08	0	1	0	0
114	817340	S	18	M	2019066992	15.8	4700	42	42	10	6	30000	1.10	120.15	78	0.6	0.2	184	118	N	42	0.08	375	0.12	N	1mm in v1,v2	N	0.08	0	1	0	0
115	817313	R	36	F	2019066975	11	13200	64	25	9	2	12000	0.50	166.98	68	0.5	0.1	177	44	CTR 0.60 Rt PE	87	0.04	426	0.12	N	nil	N	0.08	0	0	0	poor R progression
116	818825	M	26	F	2019067550	11.6	6500	66	21	10	3	22000	0.50	150.73	56	0.17	0.08	119	55	PE b/l b/l infil	41	0.08	412	0.12	N	nil	isoelectric ST in v4-v6	0.04	0	1	0	0
117	815205	S	75	M	2019066102	14	14300	92	5	2	1	140000	0.70	100.60	78	3	1.7	129	91	N	43	0.08	442	0.08	non sp	nil	N	0.08	2	1	1	0
118	818233	S	17	M	2019067333	12.5	13700	64	25	10	1	44000	0.70	165.95	68	1	0.3	120	101	b/l infil	93	0.04	413	0.12	non sp	3 mm in v2	tall T in v2-v4	0.04	0	0	0	0
119	815487	H	19	M	2019066317	18	8900	65	22	11	2	11000	0.90	134.44	72	0.5	0.11	94	38	PE b/l b/l infil	43	0.08	411	0.12	N	nil	N	0.08	0	1	0	0
120	817333	B	51	M	2019066986	15.4	5300	38	49	10	3	46000	0.80	115.89	75	0.3	0.1	95	58	N	48	0.12	450	0.12	non sp	nil	tall T in v2,v3	0.08	0	1	0	early R to S transition
121	818812	MI	40	M	2019067538	9.7	8200	61	27	10	2	60000	0.80	140.63	81	0.7	0.2	132	71	PE Rt	92	0.08	396	0.12	N	nil	N	0.06	0	0	0	0
122	818794	S	28	M	2019067525	13	9000	50	37	10	3	111000	0.70	173.33	78	0.6	0.2	167	72	N	43	0.12	460	0.12	non sp	nil	Ti in v1-v3	0.08	0	1	0	0
123	818795	I	29	M	2019067526	12.9	8100	42	32	24	2	8000	0.80	156.09	81	1.05	0.3	246	142	N	42	0.08	413	0.12	N	nil	N	0.08	0	1	0	0
124	816085	S	37	M	2019066519	16	5100	40	47	10	3	40000	0.70	167.58	82	1.1	0.45	139	71	N	87	0.12	418	0.12	non sp	1mm in v2-v6	deep S in v1-v3	0.08	0	0	0	0
125	818865	M	57	M	2019067585	14.6	6600	68	20	10	2	25000	0.60	163.31	85	0.8	0.2	168	93	N	43	0.08	434	0.12	N	nil	N	0.08	0	1	0	0
126	306345	P	21	M	2019067524	13.3	3700	63	20	10	7	41000	0.80	157.01	76	0.47	0.15	202	215	N	44	0.08	376	0.12	N	1mm in v2-v3	N	0.08	0	1	0	0
127	818633	P	50	M	2019067454	13.2	5600	65	25	10	0	22000	0.70	121.43	68	0.6	0.1	101	44	N	76	0.08	395	0.12	N	nil	N	0.08	0	0	0	0
128	816594	V	42	M	2019066735	14.7	6400	48	37	10	5	115000	1.07	99.22	78	0.9	0.3	414	267	N	46	0.12	454	0.12	N	nil	N	0.08	0	1	0	0
129	815304	V	63	F	2019066145	12.3	7500	60	27	10	3	25000	0.90	68.68	68	0.4	0.08	716	291	b/l PE	45	0.08	482	0.12	N	nil	Ti in v1-v6	0.08	0	1	0	poor R progression
130	642242	M	27	M	2019068178	10.7	3600	53	35	10	2	80000	0.90	148.23	85	1.1	0.4	78	52	N	91	0.08	415	0.12	N	nil	N	0.04	0	0	0	0
131	820234	K	72	M	201906891	10	15000	50	33	10	7	34000	1.00	64.22	68	1.2	0.5	68	55	b/l PE	46	0.08	447	0.12	N	nil	N	0.04	2	1	0	0
132	820402	K	54	F	2019068243	13.5	5400	47	41	10	2	214000	0.90	65.43	58	0.2	0.06	45	52	N	43	0.08	409	0.12	N	nil	Ti in v2,v3	0.08	0	1	0	0
133	318660	B	30	F	2019068320	12.6	2700	60	35	5	0	68000	0.50	176.61	68	0.3	0.13	125	71	N	94	0.04	427	0.12	N	nil	N	0.04	0	0	0	low voltage
134	817422	S	34	M	2019067570	11.3	13400	50	37	10	3	120000	0.70	164.05	78	0.9	0.1	92	88	N	42	0.08	418	0.12	RBBB	nil	tall T	0.06	0	1	0	0
135	820442	P	68	M	2019068274	12.6	8000	60	30	8	2	66000	1.10	65.45	72	0.4	0.2	127	66	N	57	0.08	400	0.16	N	nil	N	0.08	0	1	0	QS in v1-v3
136	819492	S	36	M	2019068201	14	3300	44	43	10	3	100000	0.08	1408.33	78	0.27	0.05	47	53	N	88	0.08	418	0.12	N	nil	N	0.08	0	0	0	0

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge													14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhthymias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
96	818907	N	N	N	P	N	55	0	N	5	0.05	861	115	180	brady	72	0.08	464	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
97	818844	N	N	N	P	N	60	0	N	5	0	100	50	160	normal	79	0.08	419	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
98	786763	N	N	N	P	N	60	0	N	5	0	100	50	170	brady	77	0.04	400	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
99	818090	N	N	N	P	N	60	0	N	5	0.07	100	30	180	brady	64	0.04	339	0.2	N	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	CLD
100	819490	N	N	N	P	N	60	0	N	5	0	170	50	180	myo, brady	62	0.08	398	0.12	N	nil	N	0.2	0	0	0	NORMAL	N	N	NIL	Normal	Nil
101	818759	N	N	N	P	N	60	0	N	5	0	150	50	170	myo, brady	94	0.04	428	P absent	N	nil	N	P absent	0	0	0	NORMAL	N	N	NIL	Normal	DM2
102	816438	N	N	N	P	N	55	0	N	5	0	170	25	180	brady	91	0.08	433	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	HTN
103	816411	N	N	N	P	N	60	0	N	6	0	170	50	180	brady	65	0.08	418	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
104	816404	N	N	N	P	N	60	0	N	7	0	160	50	180	normal	72	0.08	407	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
105	817979	N	N	N	P	N	60	0	N	6	0	170	50	160	brady	82	0.08	417	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	HTN
106	815484	N	N	N	P	N	60	0	N	7	0	170	70	180	brady	61	0.04	442	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
107	818200	N	N	N	P	N	60	0	N	5	0	150	50	180	brady	64	0.08	400	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	DM2
108	818170	N	N	N	P	N	60	0	N	5	0	170	50	180	myo	61	0.12	382	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
109	818015	N	N	N	P	N	60	0	N	5	0	170	50	170	normal	85	0.08	399	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
110 neelam	818156	N	N	N	P	N	60	0	N	5	0	170	60	190	brady	65	0.08	449	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	DM2
111 jagtar	817366	N	N	N	P	N	60	0	N	6	0	170	50	180	brady	99	0.12	448	0.12	non sp	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	HTN
112	817484	N	N	N	P	N	58	0	N	7	0	150	50	200	normal	87	0.08	399	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
113	817353	N	N	N	P	N	60	0	N	7	0	170	40	180	RBBB, brady	66	0.12	400	0.2	RBBB	nil	N	0.08	0	0	0	BBB	N	N	NIL	Normal	DM2
114	817340	N	N	N	P	N	60	0	N	5	0	170	50	210	myo, brady	83	0.08	375	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
115	817313	N	N	N	P	N	60	0	N	5	0.05	1060	76.6	480	myo	87	0.04	426	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
116	818825	N	N	N	P	N	60	0	N	5	0	150	30	270	myo , brady	75	0.08	412	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
117	815205	N	N	N	P	N	60	concentric LVH	N	5	0.05	2160	219	380	ectopics, brady	66	0.08	442	0.08	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
118	818233	N	N	N	P	global	45	0	N	5	0.05	2490	66.6	400	myo	93	0.04	413	0.12	non sp	nil	N	0.04	0	0	0	NORMAL	EF	N	NIL	Normal	CLD
119	815487	N	N	N	P	N	67	0	N	5	0.05	671	145	390	brady	66	0.08	411	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	HTN
120	817333	N	N	N	P	N	62	0	N	5	0	180	60	240	myo, brady	76	0.12	450	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
121	818812	N	N	N	P	N	62	0	N	5	0	100	60	180	normal	92	0.08	396	0.12	N	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	HTN
122	818794	N	N	N	P	N	60	0	N	5	0	150	60	180	myo, brady	74	0.12	460	0.12	non sp	nil	N	0.06	0	0	0	NORMAL	N	N	NIL	Normal	HTN
123	818795	N	N	N	P	N	62	0	N	5	0	150	60	140	brady	100	0.08	413	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
124	816085	N	N	N	P	N	58	0	N	5	0	150	45	180	myo, BBB	87	0.12	418	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
125	818865	N	N	N	N	N	62	0	N	6	0	150	60	160	brady	90	0.08	434	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
126	306345	N	N	N	P	N	62	0	N	7	0	150	60	180	myo, brady	99	0.08	376	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
127	818633	N	N	N	P	N	60	0	N	6	0	150	70	180	normal	76	0.08	395	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
128	816594	N	N	N	P	N	62	0	N	6	0	150	60	180	brady	70	0.12	454	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
129	815304	N	N	N	P	global	36	0	N	5	0.05	843	127	400	myo, brady	78	0.08	482	0.12	N	nil	N	0.08	0	0	0	NORMAL	EF	N	NIL	Normal	Nil
130	642242	N	N	N	P	N	60	0	N	5	0	200	50	180	normal	91	0.08	415	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
131	820234	N	N	N	P	N	58	0	N	5	0	150	70	170	ectopics, brady	94	0.08	447	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
132	820402	N	N	N	P	N	60	0	N	5	0.06	600	91	400	myo, brady	77	0.08	409	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
133	318660	N	N	N	P	N	60	0	N	5	0	200	50	180	myo	94	0.04	427	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
134	817422	N	N	N	P	N	60	0	N	5	0	190	50	200	BBB, brady	98	0.08	418	0.12	RBBB	nil	N	0.06	0	0	0	BBB	N	N	NIL	Normal	Nil
135	820442	N	N	N	P	N	60	0	N	30	0.15	190	27	180	myo, brady	79	0.08	400	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
136	819492	N	N	N	P	N	59	0	N	5	0	190	50	140	normal	88	0.08	418	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
137	251109	B	42	M	2019067637	15	6100	54	33	10	3	60000	1.40	76.81	79	0.4	0.07	27	22	N	59	0.08	412	0.12	RBBB	nil	isoelectric ST in v4-v6 Ti in v1-v3	0.08	0	1	0	0
138	820358	S	40	M	2019068201	14	5500	55	35	10	0	62000	0.70	138.89	70	1.2	0.5	113	53	N	55	0.2	362	0.12	LBBB	nil	1mm in v2,3 tall T in V2,3	0.08	0	2	0	0
139	821116	M	50	F	2019068518	12	4500	58	36	5	1	70000	0.60	118.65	67	1.3	0.3	115	55	N	54	0.2	404	0.12	N	nil	N	0.08	0	1	0	0
140	825378	H	68	M	2019070404	15.6	6900	65	22	10	3	36000	0.90	80.00	72	0.4	0.2	173	46	b/l PE	86	0.08	442	0.12	N	nil	N	0.04	0	0	0	QS in v1,2
141	825743	G	34	M	2019070585	15.5	9300	68	21	10	1	15000	1.30	88.33	78	1.1	0.5	654	431	N	53	0.08	455	0.12	N	nil	N	0.12	0	1	0	0
142	826614	G	26	M	2019070953	14	5500	67	22	10	1	43000	1.20	108.19	82	0.9	0.2	568	334	N	52	0.08	476	0.12	N	nil	N	0.08	0	1	0	LVH
143	826488	R	32	M	2019070862	11	4600	86	4	8	2	130000	3.90	29.62	77	8.1	7.7	402	118	PE b/l b/l infil	51	0.12	463	0.12	non sp	nil	N	0.04	0	1	0	0
144	825645	A	55	F	2019070541	11.8	8900	37	48	10	5	100000	0.50	136.47	68	0.3	0.2	162	90	N	96	0.12	471	0.12	RBBB	ST dep in v4-v6	Ti in v1-v3	0.08	0	0	0	0
145	823676	N	44	M	2019070406	12.9	4300	62	26	10	2	33000	0.90	102.22	69	0.5	0.3	108	64	N	52	0.08	391	0.12	N	nil	N	0.08	0	1	0	0
146	826360	A	60	M	2019070780	12.2	13100	84	10	5	1	150000	1.40	84.44	76	1	0.5	38	52	N	114	0.08	431	0.08	non sp	nil	N	0.04	0	0	0	0
147	824602	S	20	M	2019070111	14.2	6600	40	48	10	2	51000	1.60	45.32	78	0.5	0.2	174	97	N	73	0.08	390	0.12	N	nil	N	0.08	0	0	0	R upright in precordial leads
148	824458	J	51	M	2019069960	14.4	6100	39	43	10	8	114000	0.70	130.67	74	0.5	0.2	260	181	N	54	0.04	436	0.12	N	nil	N	0.08	0	1	0	0
149	824498	G	30	M	2019069994	12.7	5200	34	53	10	3	110000	0.70	174.60	80	0.4	0.1	109	47	N	82	0.08	428	0.12	N	nil	N	0.08	0	0	0	0
150	825536	P	27	F	2019070511	11.9	6300	86	9	4	1	226000	1.00	86.71	65	0.4	0.2	26	36	N	133	0.08	563	0.12	N	nil	ST i in all precordial	0.04	0	0	Sinus tachycardia	0
151	825243	K	17	M	2019070297	13.7	2400	55	38	5	2	80000	0.70	175.71	72	0.5	0.3	60	30	N	53	0.04	408	0.12	N	nil	N	0.16	0	1	0	0
152	825828	N	18	F	2019070641	11	3500	70	20	10	0	160000	0.50	190.12	66	0.3	0.1	45	30	N	86	0.04	438	0.12	N	nil	N	0.04	0	0	0	0
153	825631	P	18	M	2019070540	13	6900	47	40	10	3	65000	0.70	183.97	76	1.2	0.3	222	150	N	54	0.08	428	0.16	N	1mm in v2,3	N	0.04	0	1	0	0
154	825808	K	52	F	2019070626	12.8	6600	62	25	10	3	50000	0.66	136.94	87	0.8	0.4	620	201	N	80	0.04	479	0.16	N	nil	N	0.04	0	0	0	0
155	826418	MA	21	M	2019070847	13	6400	58	30	10	2	30000	0.80	140.49	68	0.45	0.13	248	110	N	92	0.08	399	0.16	N	nil	N	0.08	0	0	0	0
156	825375	N	28	M	2019070388	13.4	8500	37	50	10	3	130000	0.90	131.36	76	0.3	0.1	166	133	N	89	0.08	428	0.12	N	nil	N	0.08	0	0	0	0
157	825329	D	32	M	2019070355	13.6	7900	35	53	10	2	56000	0.77	167.53	86	0.6	0.1	190	85	N	55	0.08	416	0.16	N	nil	N	0.08	0	1	0	0
158	825313	V	42	M	2019070347	12.9	4800	40	47	10	3	90000	0.90	124.01	82	0.66	0.15	89	53	N	72	0.08	404	0.12	N	nil	N	0.08	0	0	0	0
159	824331	R	70	F	2019069861	13.7	2400	55	38	5	2	80000	0.70	#DIV/0!	72	0.5	0.3	60	30	N	88	0.04	408	0.12	N	nil	N	0.16	0	0	0	0
160	824612	J	53	F	2019070126	10.4	6100	46	42	10	2	30000	1.30	60.04	76	0.66	0.28	64	44	N	56	0.04	382	0.16	N	nil	N	0.04	0	1	0	0
161	826407	O	70	F	2019070801	12.3	11200	57	31	11	1	40000	0.14	401.39	68	0.4	0.2	175	79	N	41	0.08	388	0.12	N	nil	N	0.12	0	1	0	0
162	825741	M	32	F	2019070587	11.1	8700	45	39	10	6	90000	0.60	140.25	66	0.4	0.2	109	63	b/l PE	104	0.04	427	0.16	N	nil	N	0.04	0	0	0	0
163	826520	P	42	F	2019070886	10.8	4700	40	50	10	0	40000	0.57	113.66	56	1.2	0.6	157	78	N	64	0.08	430	0.16	N	nil	N	0.08	0	0	0	0
164	826472	L	49	M	2019070858	15.2	5000	64	23	10	3	11000	1.13	87.24	78	0.2	0.1	75	28	N	57	0.08	399	0.12	N	nil	tall T in v2-v5	0.04	0	1	0	0
165	825969	S	30	M	2019070681	15.7	5300	51	38	10	1	33000	0.50	238.33	78	0.4	0.1	229	119	N	68	0.04	401	0.12	N	1mm in v2,v3	tall T in v2-v6	0.08	0	0	0	0
166	826508	R	52	M	2019070892	13.7	4100	64	21	10	5	54000	0.80	125.28	82	0.3	0.1	250	168	b/l PE	72	0.08	412	0.12	N	nil	N	0.08	0	0	0	0
167	825811	D	31	M	2019070623	14.6	8000	60	30	8	2	64000	0.53	242.79	85	0.8	0.5	552	247	cardiomegaly	58	0.12	442	0.12	non sp	nil	convex ST in v1,2,3	0.12	0	1	0	0
168	826166	S	34	M	2019070738	14.2	1100	31	57	10	2	32000	0.90	125.96	77	0.6	0.37	192	161	N	87	0.08	428	0.16	non sp	nil	N	0.04	0	0	0	0
169	826493	H	28	M	2019070868	14.1	6500	46	50	2	2	24000	0.60	202.22	78	0.44	0.11	282	260	N	79	0.08	376	0.12	N	2mm in v2	N	0.08	0	0	0	0
170	826538	S	27	M	2019070906	12.2	6500	49	39	10	2	24000	0.90	132.53	76	0.9	0.6	144	65	N	59	0.08	421	0.16	N	nil	QS in v1-3	isoelectric P	0	1	0	0
171	826389	A	21	M	2019070794	15.3	18800	32	56	10	2	22000	0.71	162.95	70	0.6	0.4	333	202	N	98	0.08	413	0.12	N	nil	N	0.04	0	0	0	0
172	826449	S	46	M	2019070822	13.4	6900	56	30	10	4	70000	0.80	127.29	78	0.6	0.2	119	61	N	57	0.08	408	0.12	N	1mm in v2,3	hyperacute in v3,v4	0.08	0	1	0	0
173	825423	K	20	M	2019070441	15.6	7800	46	40	10	4	66000	0.80	145.83	70	0.5	0.2	101	55	N	94	0.08	414	0.12	N	nil	U wave	0.12	0	0	0	0
174	825424	A	17	F	2019070440	12.9	5900	34	53	10	3	30000	0.50	180.06	62	0.3	0.2	77	38	N	101	0.08	404	0.12	N	nil	Ti in v1-v3	0.04	0	0	0	0
175	826022	P	45	M	2019070691	10.5	3200	75	15	10	0	14000	0.70	147.02	78	0.3	0.2	88	39	N	52	0.04	393	0.12	N	nil	N	0.08	0	1	0	0
176	825715	J	19	M	2019070570	13.2	6100	28	60	10	2	198000	0.40	285.69	68	8	6.6	5600	5630	N	66	0.08	446	0.12	N	nil	N	0.16	0	0	0	0
177	826256	P	38	M	2019070764	14.9	7400	43	47	10	0	90000	0.80	138.13	78	0.6	0.3	304	179	PE R/t	96	0.08	410	0.16	N	nil	N	0.04	0	0	0	0
178	824584	R	24	M	2019070095	14.2	5500	58	29	10	3	27000	1.02	110.57	70	0.6	0.4	208	102	N	55	0.04	367	0.12	N	nil	N	0.12	0	1	0	0
179	824582	C	54	M	2019070098	10.6	9100	62	25	10	3	71000	2.30	39.47	76	4.7	3.9	8137	3589	N	55	0.08	456	0.12	N	nil	N	0.04	0	1	0	0
180	823292	S	43	F	2019069460	11.9	2300	30	58	10	2	62000	0.86	86.55	65	0.5	0.12	110	52	b/l infil	89	0.08	414	0.12	N	nil	N	0.08	0	0	0	0
181	824367	A	48	F	2019069904	11	1100	29	57	10	4	22000	0.80	95.03	70	0.3	0.2	65	48	b/l infil B/l PE	54	0.08	448	0.12	N	nil	N	0.04	0	1	0	0
182	825346	A	47	M	2019070698	16.2	7000	70	17	10	3	30000	0.80	140.47	87	0.5	0.2	28	29	N	62	0.08	405	0.12	N	1mm in v1-v3	tall T in v2-v6	0.08	0	0	0	0
183	842654	N	33	M	2019078141	10.5	3400	60	26	10	4	29000	1.60	45.36	78	1.2	0.67	157	128	N	112	0.08	431	0.12	N	nil	N	0.08	0	0	0	Q3T3 IN III

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge													14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhthymias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
137	251109	N	N	N	P	N	60	0	N	5	0	190	30	140	RBBB, myo, brady	99	0.08	412	0.12	RBBB	nil	N	0.04	0	0	0	BBB	N	N	NIL	Normal	CKD
138	820358	N	N	N	P	N	60	0	N	5	0	190	50	150	myo, LBBB, brady	64	0.2	362	0.12	LBBB	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
139	821116	N	N	N	P	N	60	0	N	6	0	190	25	169	brady	68	0.2	404	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
140	825378	N	N	N	P	N	58	0	N	7	0	190	50	170	myo	86	0.08	442	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
141	825743	N	N	N	P	N	60	0	N	5	0.11	1960	500	500	brady	100	0.08	455	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	CKD
142	826614	N	N	N	P	N	60	0	N	5	0	100	25	150	LVH, brady	96	0.08	476	0.12	N	nil	N	0.08	0	0	0	LVH	N	LVH	NIL	LVH	CLD
143	826488	N	N	N	P	N	60	0	N	20	0.8	3090	96.2	400	BBB, brady	74	0.12	463	0.12	N	nil	N	0.04	0	0	0	NORMAL	BBB	N	NIL	Normal	CKD
144	825645	N	N	N	P	N	60	0	N	5	0	150	30	230	RBBB, myo	96	0.12	471	0.12	RBBB	nil	N	0.08	0	0	0	BBB	N	N	NIL	Normal	Nil
145	823676	N	N	N	P	N	60	0	N	6	0	150	30	230	brady	83	0.08	391	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
146	826360	N	N	N	N	N	60	0	N	6	0	150	30	230	BBB	94	0.08	431	0.08	N	nil	N	0.04	0	0	0	NORMAL	BBB	N	NIL	Normal	Nil
147	824602	N	N	N	P	N	60	0	N	7	0	150	25	170	normal	73	0.08	390	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
148	824458	N	N	N	P	N	60	0	N	8	0	150	30	230	brady	80	0.04	436	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
149	824498	N	N	N	P	N	60	0	N	8	0	150	30	230	normal	82	0.08	428	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
150	825536	N	N	N	P	N	58	0	N	9	0	150	40	230	tachy	95	0.08	563	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
151	825243	N	N	N	P	N	60	0	N	5	0	150	30	140	brady	88	0.04	408	0.12	N	nil	N	0.16	0	0	0	NORMAL	N	N	NIL	Normal	Nil
152	825828	N	N	N	P	N	60	0	N	5	0.5	408	64.8	230	myo	86	0.04	438	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
153	825631	N	N	N	P	N	60	0	N	5	0	180	40	230	myo, brady	92	0.08	428	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
154	825808	N	N	N	P	N	58	0	N	5	0	180	40	150	normal	80	0.04	479	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
155	826418	N	N	N	P	N	60	0	N	5	0	180	25	230	normal	92	0.08	399	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
156	825375	N	N	N	P	N	60	0	N	5	0	180	40	230	normal	89	0.08	428	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
157	825329	N	N	N	P	N	60	0	N	5	0	180	40	160	brady	97	0.08	416	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
158	825313	N	N	N	P	N	55	0	N	6	0	180	25	230	normal	72	0.08	404	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
159	824331	N	N	N	P	N	60	0	N	5	0	150	30	140	normal	88	0.04	408	0.12	N	nil	N	0.16	0	0	0	NORMAL	N	N	NIL	Normal	Nil
160	824612	N	N	N	P	N	60	0	N	6	0	180	40	230	brady	71	0.04	382	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CKD
161	826407	N	N	N	P	N	60	0	N	5	0	180	40	170	brady	65	0.08	388	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
162	825741	N	N	N	P	N	60	0	N	8	0	180	40	230	normal	99	0.04	427	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
163	826520	N	N	N	P	N	62	0	N	9	0	180	40	230	normal	64	0.08	430	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
164	826472	N	N	N	P	N	60	0	N	10	0	180	40	140	myo, brady	86	0.08	399	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
165	825969	N	N	N	P	N	60	0	N	7	0	180	40	230	myo	68	0.04	401	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
166	826508	N	N	N	P	N	58	0	N	7	0	180	25	230	normal	72	0.08	412	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CLD
167	825811	N	N	N	P	N	60	0	N	5	0	180	40	130	myo, brady	90	0.12	442	0.12	non sp	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
168	826166	N	N	N	P	N	56	0	N	9	0	180	40	230	normal	87	0.08	428	0.16	non sp	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
169	826493	N	N	N	P	N	60	0	N	11	0	180	40	140	myo	79	0.08	376	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
170	826538	N	N	N	P	N	60	0	N	5	0	180	26	230	myo, brady	100	0.08	421	0.16	N	nil	N	isoelectric P	0	0	0	NORMAL	N	N	NIL	Normal	Nil
171	826389	N	N	N	P	N	58	0	N	5	0	180	30	230	normal	98	0.08	413	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CLD
172	826449	N	N	N	P	N	60	0	N	5	0	170	40	230	myo, brady	73	0.08	408	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
173	825423	N	N	N	P	N	60	0	N	5	0	170	40	150	myo	94	0.08	414	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
174	825424	N	N	N	P	N	60	0	N	7	0	160	40	230	myo	98	0.08	404	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
175	826022	N	N	N	P	N	60	0	N	9	0	150	50	230	brady	76	0.04	393	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
176	825715	N	N	N	P	N	60	0	N	12	0	100	40	230	normal	66	0.08	446	0.12	N	nil	N	0.16	0	0	0	NORMAL	N	N	NIL	Normal	Nil
177	826256	N	N	N	P	N	66	0	N	6	0	80	60	230	normal	96	0.08	410	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
178	824584	N	N	N	P	N	60	0	N	7	0	70	40	170	brady	62	0.04	367	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
179	824582	N	N	N	P	N	60	0	N	5	0.05	2200	500	230	myo, brady	95	0.08	456	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CKD
180	823292	N	N	N	P	N	60	0	N	5	0	70	40	200	normal	89	0.08	414	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
181	824367	N	N	N	P	N	60	0	N	5	0	60	45	230	brady	83	0.08	448	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
182	825346	N	N	N	P	N	55	0	N	9	0	100	45	230	myo	62	0.08	405	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
183	842654	N	N	N	P	N	60	0	N	7	0	110	45	230	BBB	100	0.08	431	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
184	780929	S	70	F	2019079201	9.5	2600	63	22	10	5	92000	3.00	16.53	60	0.19	0.06	73	45	N	52	0.08	437	0.16	non sp	nil	N	0.08	0	1	0	Q in v4-v6
185	845040	H	55	M	2019079235	12.3	3400	60	32	5	3	74000	0.70	128.17	76	1.1	0.56	435	228	N	79	0.08	447	0.12	N	nil	N	0.04	0	0	0	0
186	845137	R	43	M	2019079317	15.6	5900	39	50	10	1	59000	0.50	199.39	74	0.5	0.1	184	144	N	58	0.08	382	0.12	non sp	nil	N	0.08	0	1	0	0
187	845193	J	38	F	2019079356	10.1	4900	37	53	7	3	60000	0.50	156.54	65	0.4	0.2	187	178	Rt PE	53	0.04	412	0.16	N	nil	N	0.04	0	1	0	Q3 in v1-v3
188	845195	M	60	F	2019079352	13.3	5900	38	50	10	2	59000	1.60	50.46	66	0.5	0.2	346	261	N	87	0.04	455	0.16	N	nil	N	0.04	0	0	0	0
189	847493	I	38	M	2019080404	12.7	7600	63	28	9	0	80000	0.70	157.86	78	0.7	0.1	31	48	N	54	0.08	422	0.16	N	nil	tall T in v2-v4	0.08	0	1	0	0
190	847374	SL	71	M	2019080336	12.4	3300	58	30	10	2	12000	1.08	57.68	65	1.6	0.5	143	71	Rt PE	84	0.08	458	0.08	N	nil	N	0.04	0	0	0	0
191	847347	J	55	M	2019080332	12.8	8900	68	20	10	2	26000	0.79	107.59	72	0.8	0.3	1012	480	b/l infil B/l PE	54	0.08	441	0.08	N	nil	tall T in v2-v4	0.04	0	1	0	0
192	844279	D	60	F	2019078879	11.4	2000	80	15	5	0	92000	3.90	16.47	68	0.3	0.1	79	82	b/l infil B/l PE	80	0.08	520	0.16	non sp	nil	Ti in all precordial I, II,III, avF	0.04 in avR, avL	0	0	0	0
193	846537	M	62	F	2019079980	11.5	14200	86	8	5	1	70000	0.90	67.53	66	0.6	0.1	241	83	b/l infil B/l PE	53	0.12	473	0.12	RBBB	nil	Ti in v1	0.08	0	1	0	0
194	846736	P	34	F	2019080153	13.1	12900	50	35	15	0	19000	2.30	38.63	71	0.9	0.5	591	268	N	184	0.08	437	absent P	non sp	nil	ST I in all precordial Ti in v1-v6, I, II,III, Avf	Ti	0	0	SVT	0
195	846615	R	52	M	2019080050	12.6	12800	75	20	4	1	50000	0.80	100.23	76	0.9	0.2	42	35	B/l Nodules	107	0.08	456	0.12	N	nil	N	0.08	0	0	0	0
196	846931	R	50	M	2019080227	9.6	2600	57	30	10	3	122000	1.60	58.06	75	0.3	0.1	65	32	N	54	0.08	458	0.16	N	nil	N	0.08	0	1	0	0
197	846583	H	58	M	2019080022	10.4	3000	60	28	10	2	15000	0.70	130.16	80	1.96	1.4	214	154	N	88	0.08	432	0.16	N	nil	N	0.04	0	0	0	0
198	846058	S	25	M	2019080026	13.6	6700	40	46	11	3	93000	0.60	207.64	78	0.9	0.3	134	85	N	58	0.08	345	0.16	N	Imm in v2,3,4	tall T in v2-5	0.08	0	1	0	0
199	846645	R	10	M	2019080090	12.9	6300	65	24	9	2	150000	0.40	180.56	40	4.9	4.1	365	380	N	56	0.08	346	0.16	N	nil	N	0.08	0	1	0	0
200	847841	H	20	M	2019030665	16.5	2200	50	38	10	2	46000	0.50	243.33	73	0.7	0.2	45	25	N	70	0.08	367	0.16	N	1mm in v2-v6	N	0.08	0	0	0	0
201	847656	V	48	M	2019080666	13.3	6300	54	34	10	2	18000	0.73	136.53	78	0.8	0.5	196	96	N	70	0.08	365	0.16	N	nil	N	0.08	0	0	0	0
202	846765	G	31	M	2019080190	11.7	5400	62	26	10	2	125000	0.70	162.20	75	0.4	0.1	39	42	N	46	0.08	410	0.2	N	nil	N	0.04	0	2	0	S1Q3+
203	846551	U	16	M	2019079994	13.4	2600	42	43	10	5	81000	0.52	225.21	68	0.22	0.14	61	38	N	58	0.08	436	0.08	N	nil	N	0.08	0	1	0	0
204	846601	S	62	M	2019080045	13.7	5300	50	45	4	1	50000	1.17	68.52	74	0.8	0.12	203	104	N	118	0.08	446	0.12	N	nil	N	0.08	0	0	0	0
205	846587	LD	24	M	2019080127	14	6300	43	41	14	2	37000	0.90	139.63	78	1.12	0.3	109	31	N	76	0.08	399	0.12	N	nil	N	0.08	0	0	0	0
206	846761	S	52	F	2019080186	12.3	1400	48	40	10	2	40000	0.60	117.74	68	0.5	0.17	138	71	N	59	0.08	425	0.16	N	nil	N	0.08	0	1	0	0
207	845762	K	38	F	2019079606	14	5500	47	40	10	3	11000	0.80	103.86	69	1.01	0.38	425	190	N	103	0.08	424	0.08	N	nil	N	0.08	0	0	0	0
208	845758	S	60	F	2019079598	13	8200	35	50	13	2	45000	1.60	32.47	55	1.02	0.5	227	167	N	57	0.08	414	0.16	N	nil	tall T lin v1-v6	0.08	0	1	0	0
209	847570	S	60	F	2019080493	10.6	3300	80	13	6	1	100000	2.09	29.37	65	0.7	0.15	215	152	N	56	0.08	434	0.16	N	nil	N	0.08	0	1	0	0
210	840377	K	45	F	2019079864	12.4	3400	46	42	8	4	85000	0.60	102.81	55	0.5	0.14	124	80	N	84	0.04	440	0.12	N	nil	N	0.04	0	0	0	0
211	846519	G	26	M	2019079959	15.3	4300	30	55	10	5	31000	0.80	162.29	82	1.2	0.3	313	212	N	52	0.08	432	0.12	N	nil	N	0.04	0	1	0	0
212	846392	T	34	M	2019079880	13.4	7000	58	30	10	2	40000	0.70	159.84	76	1.1	0.7	161	109	N	98	0.04	408	0.12	N	nil	N	0.04	0	0	0	0
213	845772	V	53	M	2019079615	14.4	6800	64	23	10	3	94000	1.50	92.12	77	0.5	0.1	443	87	Rt PE	55	0.08	442	0.12	N	nil	N	0.04	0	1	0	0
214	846706	V	18	M	2019080130	11.4	7300	68	20	10	2	14000	0.90	128.02	68	1.2	0.7	1180	489	N	106	0.04	404	0.12	N	nil	N	0.04	0	0	0	0
215	842078	V	39	M	2019077992	4.8	6300	60	27	10	3	40000	0.70	130.26	65	0.3	0.1	74	21	Rt PE	51	0.08	382	0.12	N	nil	N	0.08	0	1	0	0
216	301609	H	40	M	2019079621	13.4	11700	61	27	10	2	30000	0.85	133.99	82	0.7	0.2	246	152	B/l infil	65	0.12	471	0.16	N	nil	N	0.08	0	0	0	0
217	840915	P	57	F	2019077367	9.1	11500	66	21	10	3	130000	1.60	35.52	58	5.2	4.8	69	46	B/l infil	50	0.12	450	0.16	N	nil	N	0.08	0	1	0	0
218	845111	C	55	M	2019079290	11.9	6300	61	25	10	4	60000	1.07	79.44	72	0.8	0.2	231	163	b/l PE	110	0.08	438	0.12	N	nil	N	0.04	0	0	0	0
219	841027	A	23	M	2019077473	12.9	17000	94	3	3	0	21000	3.70	31.62	72	2.2	0.6	130	22	b/l PE b/l nodules	114	0.08	424	0.16	N	nil	tall T in v2,3	0.08	0	0	0	0
220	845724	P	31	F	2019079566	10.2	10900	45	44	10	1	11000	0.70	143.39	78	0.7	0.1	181	136	N	96	0.04	499	0.08	N	nil	N	0.08	0	0	0	0
221	843380	J	23	F	2019078579	9.4	3700	66	25	5	4	16000	0.50	160.23	58	0.5	0.1	623	480	N	90	0.04	423	0.12	N	nil	N	0.08	0	0	0	0
222	215722	N	57	F	2019079822	11.5	7100	34	52	10	4	58000	0.80	75.94	62	0.3	0.1	169	52	PE lt	87	0.04	419	0.08	N	nil	N	0.04	0	0	0	0
223	840116	B	35	F	2019077079	10.6	11800	66	22	10	2	34000	0.80	108.46	70	1.08	0.53	2483	595	PE b/l	105	0.08	403	0.12	N	nil	N	0.04	0	0	0	0
224	846439	B	37	M	2019079913	16	2600	53	33	10	4	30000	0.90	123.98	78	0.6	0.1	219	136	N	73	0.04	422	0.16	N	nil	N	0.04	0	0	0	0
225	846767	S	24	M	2019080191	15	7700	41	47	10	2	120000	0.50	232.00	72	0.8	0.5	381	180	N	77	0.08	410	0.12	N	nil	tall T in v2,3	0.08	0	0	0	0
226	847589	R	46	M	2019080512	15	5600	33	52	12	3	28000	0.65	156.67	78	0.8	0.2	190	91	N	70	0.08	361	0.12	N	nil	N	0.08	0	0	0	0
227	846755	C	27	M	2019080178	14	5700	41	44	12	3	41000	0.80	141.25	72	1.02	0.2	111	54	N	85	0.04	396	0.12	N	nil	N	0.04	2	0	0	0
228	847617	R	30	M	2019080545	15	1900	59	30	10	1	22000	1.00	116.11	76	0.5	0.1	246	185	N	91	0.08	404	0.12	N	nil	N	0.08	0	0	0	0
229	846708	N	54	M	2019080132	16	2500	47	40	10	3	30000	0.80	104.51	70	0.4	0.1	40	54	N	88	0.08	415	0.12	N	nil	N	0.08	0	0	0	0
230	846280	S	32	M	2019079825	14	5600	42	45	10	3	65000	0.70	162.86	76	0.3	0.1	89	85	N	58	0.04	401	0.12	N	nil	N	0.08	0	1	0	0
231	847511	D	65	F	2019080444	11	11200	72	20	8	0	16000	0.80	75.26	68	1.3	0.7	523	284	Rt PE	96	0.04	438	0.16	N	nil	N	0.08	0	0	0	Qs in vi,2
232	846099	G	71	M	2019079770	6.7	11900	63	20	11	6	64000	1.50	41.53	65	1	0.7	613	449	PE b/l	92	0.04	454	0.16	N	nil	N	0.08	0	0	0	Qs in v1-v3 Qs in III,Avf

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge												14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhthymias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
184	780929	N	N	N	P	N	60	0	N	7	0	120	30	230	brady	99	0.08	437	0.16	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
185	845040	N	N	N	P	N	60	0	N	12	0	140	45	230	normal	79	0.08	447	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CLD
186	845137	N	N	N	P	N	60	0	N	5	0	80	45	140	brady	67	0.08	382	0.12	non sp	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
187	845193	N	N	N	P	N	62	0	N	7	0	50	25	230	myo, brady	70	0.04	412	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
188	845195	N	N	N	P	N	60	0	N	8	0	80	45	230	normal	87	0.04	455	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CLD
189	847493	N	N	N	P	N	60	0	N	8	0	90	45	230	myo, brady	96	0.08	422	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
190	847374	N	N	N	P	N	58	0	N	5	0	110	45	200	normal	84	0.08	458	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
191	847347	N	N	N	P	N	60	0	N	11	0	80	45	230	myo, brady	72	0.08	441	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
192	844279	P	N	N	P	global	45	0	N	31	0.1	3350	500	230	myo	80	0.08	520	0.16	non sp	nil	N	0.04 in avR, avL	0	0	0	NORMAL	EF	N	NIL	Normal	CKD
193	846537	N	N	N	P	N	62	0	N	5	0.02	4430	500	230	myo, brady	100	0.12	473	0.12	RBBB	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
194	846736	N	N	N	P	N	62	0	N	5	0.05	930	72.4	230	myo, tachy	98	0.08	437	absent P	non sp	nil	N	Ti	0	0	0	NORMAL	N	N	NIL	Normal	CKD
195	846615	N	N	N	P	N	62	0	N	5.9	0.22	3010	411	200	normal	99	0.08	456	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
196	846931	N	N	N	P	N	62	0	N	11	0	180	35	230	brady	73	0.08	458	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
197	846583	N	N	N	P	N	62	0	N	5	0	180	35	230	normal	88	0.08	432	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
198	846058	N	N	N	P	N	62	0	N	5	0	180	35	180	myo, brady	64	0.08	345	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
199	846645	N	N	N	P	N	62	0	N	6	0	180	35	230	myo, brady	78	0.08	346	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
200	847841	N	N	N	P	N	60	0	N	8	0	180	35	190	normal	70	0.08	367	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
201	847656	N	N	N	P	N	62	0	N	6	0	180	35	230	normal	68	0.08	365	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
202	846765	N	N	N	P	N	62	0	N	5	0	180	35	210	brady	98	0.08	410	0.2	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
203	846551	N	N	N	P	N	55	0	N	5	0	180	35	150	brady	74	0.08	436	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
204	846601	N	N	N	P	N	62	0	N	5	0	180	35	210	normal	98	0.08	446	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
205	846587	N	N	N	P	N	62	0	N	5	0	180	35	200	normal	76	0.08	399	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
206	846761	N	N	N	P	N	62	0	N	6	0	180	35	210	brady	76	0.08	425	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
207	845762	N	N	N	P	N	55	0	N	6	0	180	35	170	normal	96	0.08	424	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
208	845758	N	N	N	P	N	62	0	N	7	0	180	35	210	myo, brady	76	0.08	414	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
209	847570	N	N	N	P	N	62	0	N	5	0.5	2690	181	400	brady, myo	69	0.08	434	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
210	840377	N	N	N	P	N	60	0	N	8	0	170	26	210	normal	84	0.04	440	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
211	846519	N	N	N	P	N	62	0	N	5	0	190	27	160	brady	96	0.08	432	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
212	846392	N	N	N	P	N	62	0	N	9	0	180	28	210	normal	98	0.04	408	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
213	845772	N	N	N	P	N	55	0	N	5	0.5	870	168	350	myo, brady	99	0.08	442	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
214	846706	N	N	N	P	N	60	0	N	10	0	100	30	210	normal	100	0.04	404	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
215	842078	N	N	N	P	N	62	0	N	11	0	100	36	170	brady	71	0.08	382	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
216	301609	N	N	N	P	N	60	0	N	6	0.4	200	66	210	normal	66	0.12	471	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
217	840915	N	N	N	P	N	62	0	N	5	0.05	200	80	220	brady	62	0.12	450	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
218	845111	N	N	N	P	N	62	0	N	11	0	100	30	220	normal	98	0.08	438	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
219	841027	N	N	N	P	N	62	0	N	5	0.08	4090	362	400	myo	99	0.08	424	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
220	845724	N	N	N	P	N	62	0	N	12	0	100	40	220	normal	96	0.04	499	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
221	843380	N	N	N	P	N	55	0	N	11	0	200	40	220	normal	90	0.04	423	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
222	215722	N	N	N	P	N	62	0	N	5	0	200	60	160	normal	87	0.04	419	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
223	840116	N	N	N	P	N	62	0	N	5	0	200	40	220	normal	100	0.08	403	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
224	846439	N	N	N	N	N	58	0	N	5	0	200	40	220	normal	73	0.04	422	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
225	846767	N	N	N	P	N	62	0	N	5	0	200	40	170	myo	77	0.08	410	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
226	847589	N	N	N	P	N	62	0	N	6	0	200	30	220	normal	70	0.08	361	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
227	846755	N	N	N	P	N	55	0	N	5	0	200	40	220	ectopics	85	0.04	396	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
228	847617	N	N	N	P	N	62	0	N	7	0	200	40	150	normal	91	0.08	404	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
229	846708	N	N	N	P	N	62	0	N	5	0	200	40	220	normal	88	0.08	415	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
230	846280	N	N	N	P	N	58	0	N	5	0	200	50	220	brady	61	0.04	401	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
231	847511	N	N	N	P	N	62	0	N	5	0	200	40	140	myo	96	0.04	438	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
232	846099	N	N	N	P	N	62	0	N	5	0.05	1140	159	450	myo	92	0.04	454	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD

Sr No	MRD	NAME	AGE	GENDER	ADMISSION NO	HB	TLC	DLC (N)	DLC (L)	DLC (E)	DLC (B)	PLT	CR	CRCL	Body wt	BILIRUBIN T	BILIRUBIN D	OT	PT	CXR	HR	QRS	QTc	PR INTERVAL	Type of BBB	ST ELEVATION	ST /T WAVE CHANGES	Tpe	Ectopics 0-None 1-APC 2-VPC	Bradyarrthymias 0-none 1-sinus bradycardia 2-1st degree 3-2nd degree 4-complete	Tachyarrthymias 0-none 1-AF 2-VT 3-VF 4-TdP	MISC
233	848035	K	45	F	2019080755	11.7	3400	70	20	10	0	16000	0.60	127.11	68	1.2	1	5702	1827	N	110	0.08	445	0.12	N	nil	ST i in v4-v6 Ti in v2,4,6	0.08	2	0	0	0
234	843908	G	30	M	2019078605	14	11400	33	49	10	8	63000	1.10	100.00	72	0.6	0.1	29	43	N	107	0.08	378	0.12	N	nil	N	0.04	0	0	0	0
235	846585	A	84	M	2019080031	14	4900	57	29	10	4	49000	1.20	42.78	66	0.3	0.1	123	54	N	39	0.08	385	0.2	N	nil	tall T in v4-6	0.08	0	2	0	0
236	809616	S	40	F	2019063928	14	15800	44	45	11	0	28000	1.20	69.85	71	4.4	3.5	145	130	PE b/l	109	0.04	492	0.16	N	nil	Ti in all precordials	0.04	0	0	0	0
237	810658	L	38	F	2019064388	8.9	3700	51	39	10	0	213000	0.20	415.44	69	0.6	0.5	80	56	Cardiomegaly b/l PE b/l Infil	73	0.06	517	0.16	N	nil	Ti in v1-v3	0.08	0	0	0	0
238	810590	A	50	M	2019064334	16	2400	80	20	0	0	26000	1.70	51.47	70	2.2	1.8	2931	1632	N	127	0.08	432	0.12	N	nil	Tall T in v1-v3	0.04	0	0	1	0
239	813082	M	25	M	2019065311	14	9900	65	20	10	5	40000	0.70	164.29	72	0.9	0.1	127	50	N	85	0.08	415	0.12	N	nil	N	0.04	0	0	0	0
240	807257	PL	73	F	2019063002	10.3	8200	57	30	10	3	100000	0.75	63.28	60	2.1	1.7	88	45	PE b/l b/l Infil	81	0.08	511	0.12	N	nil	isoelectric ST in v4-v6	0.08	0	0	0	poor R prog
241	808880	S	32	M	2019063651	10.5	18300	43	48	8	1	30000	1.40	77.14	72	5.6	3.3	171	73	Cardiomegaly b/l PE b/l Infil	100	0.08	446	0.08	N	nil	N	0.04	0	0	0	0
242	810651	A	19	M	2019064382	14	4900	47	40	10	3	22000	0.90	130.71	70	1.2	0.3	41	28	N	112	0.08	429	0.12	N	nil	N	0.04	0	0	0	0
243	811494	P	25	F	2019064651	9	3000	76	14	8	2	10000	0.70	131.88	68	0.6	0.1	30	19	N	69	0.04	425	0.12	N	nil	Ti in v1-v3	0.08	0	0	0	0
244	767657	A	31	M	2019064890	16	9300	71	14	10	5	220000	0.80	143.82	76	0.6	0.1	50	39	N	75	0.08	433	0.12	N	nil	N	0.08	0	0	0	0
245	812361	B	50	M	2019065064	12	7800	65	23	10	2	28000	0.80	121.88	78	0.5	0.2	54	32	N	50	0.08	364	0.12	N	nil	N	0.04	0	1	0	0
246	797689	K	53	M	2019064951	12.5	2500	65	22	10	3	140000	1.30	73.43	79	0.9	0.2	44	22	N	67	0.08	437	0.16	N	nil	N	0.08	0	0	0	0
247	811186	K	19	M	2019065195	14.3	9300	29	58	7	6	70000	0.60	201.67	72	0.5	0.1	98	58	N	66	0.08	413	0.16	N	P	2mm in v2-v6	0.08	0	0	0	0
248	812233	K	52	F	2019064972	11.6	4600	35	50	10	5	53000	0.80	103.89	80	0.75	0.5	141	66	N	68	0.08	443	0.12	N	nil	isoelectric ST in v4-v6	iso	0	0	0	0
249	809747	SS	55	M	2019064004	10.1	4700	43	41	10	6	60000	2.10	44.41	79	0.5	0.3	178	88	PE b/l b/l Infil	150	0.04	388	AF	N	nil	N	0.08	0	0	1	0
250	811746	R	76	F	2019064835	10.9	3100	44	43	10	3	128000	0.70	71.24	66	0.2	0.1	53	23	N	63	0.04	415	0.12	N	nil	N	0.04	0	0	0	0
251	811491	L	27	M	2019064647	14	3000	57	31	10	2	40000	0.80	143.21	73	0.5	0.1	140	58	N	75	0.08	385	0.12	N	nil	N	0.08	0	0	0	0
252	812212	H	55	M	2019064931	15	4100	72	17	10	1	20000	0.70	114.68	68	0.6	0.1	307	152	N	71	0.08	469	0.16	N	nil	N	0.08	0	0	0	0
253	811563	A	28	M	2019064705	15	5000	47	38	10	5	67000	0.90	124.44	72	0.7	0.3	200	142	N	78	0.04	385	0.12	N	nil	N	0.04	0	0	0	0
254	811618	R	51	M	2019064754	13.4	1900	64	24	11	1	50000	0.90	109.88	80	0.46	0.1	112	93	N	85	0.08	422	0.12	N	nil	N	0.08	0	0	0	0
255	810611	S	25	M	2019064335	12	2800	65	23	11	1	68000	0.70	164.29	72	0.5	0.2	118	87	N	78	0.08	456	0.12	N	nil	N	0.08	0	0	0	0
256	811593	A	32	F	2019064722	13	12100	72	17	10	1	22000	0.60	140.25	66	0.4	0.1	58	45	N	65	0.04	427	0.16	N	nil	N	0.08	0	0	0	0
257	814561	R	23	M	2019032657	15.4	7300	51	37	10	2	30000	0.70	181.07	78	0.6	0.2	54	35	N	62	0.04	328	0.12	N	nil	tall T in v3-v6	0.08	0	0	0	0
258	813808	P	17	F	2019065608	10.2	3800	60	28	10	2	35000	0.70	114.09	55	1.6	0.2	132	48	PE b/l b/l Infil	86	0.04	442	0.12	N	nil	N	0.08	0	0	0	0
259	815317	S	48	F	2019066153	12.7	19800	70	21	9	0	45000	0.76	95.75	67	1.98	1.4	10096	3969	N	62	0.04	396	0.12	N	nil	N	0.08	0	0	0	0
260	814677	RK	18	M	2019065958	11.4	4300	52	39	8	1	70000	0.80	148.26	70	1.3	0.3	190	180	N	61	0.08	425	0.16	N	nil	tall T in v4-v6	0.04	0	0	0	0
261	813854	R	52	M	2019065634	14.5	8600	73	17	9	1	82000	0.76	131.87	82	0.7	0.2	120	80	N	113	0.08	425	0.16	N	nil	N	0.04	0	0	0	0
262	813766	S	39	F	2019065590	9.9	13500	31	61	8	0	135000	0.80	101.35	68	6.2	6	181	90	PE b/l b/l Infil	106	0.08	488	0.08	N	nil	N	0.04	0	0	0	0
263	813888	N	37	M	2019065661	13.4	4600	58	29	10	3	100000	0.69	153.42	74	0.2	0.14	85	46	N	76	0.04	398	0.12	N	nil	N	0.04	0	0	0	0
264	815404	S	42	M	2019066212	14.3	8600	40	47	10	3	70000	0.80	132.71	78	0.7	0.39	204	147	N	88	0.08	432	0.12	N	nil	N	0.08	0	0	0	0
265	815439	A	50	M	2019066266	16.5	4000	61	26	10	3	36000	0.70	139.29	78	1.04	0.7	194	174	N	76	0.08	440	0.12	N	nil	N	0.08	0	0	0	0
266	815243	J	50	M	2019066067	13.5	7700	60	27	10	3	195000	0.79	120.25	76	1.3	0.2	44	37	b/l Infil	64	0.08	403	0.16	N	nil	N	0.04	0	0	0	0
267	311952	A	32	M	2019066134	15.4	10800	33	54	10	3	40000	0.90	136.67	82	0.74	0.39	508	479	N	102	0.08	416	0.12	N	2mm in v2	tall T in v2,3	0.08	0	0	0	0
268	815275	P	27	M	2019066136	13.9	3100	42	45	10	3	90000	1.00	106.72	68	0.8	0.1	118	110	N	97	0.08	433	0.12	N	nil	N	0.04	0	0	0	0
269	815224	S	30	M	2019066112	13.6	3500	33	51	10	6	41000	0.80	141.32	74	0.7	0.2	190	69	N	61	0.04	358	0.2	N	nil	N	0.08	0	0	0	0
270	814913	R	44	M	2019066051	11.4	16000	84	10	5	1	155000	0.76	147.37	84	0.6	0.2	20	18	N	72	0.04	442	0.12	N	nil	N	0.08	0	0	0	0
271	817054	K	36	M	2019065931	13.9	8600	80	8	10	2	216000	0.38	315.50	83	1.2	0.3	33	23	N	96	0.04	435	0.12	N	nil	N	0.12	0	0	0	0
272	815286	P	45	M	2019066138	13	5600	45	47	6	2	14000	0.60	171.53	78	0.5	0.22	179	70	N	87	0.04	424	0.12	N	nil	N	0.08	0	0	0	0
273	614527	R	37	M	2019065146	11.4	6000	56	33	10	1	82000	1.00	103.00	72	0.55	0.15	95	102	N	104	0.04	412	0.08	N	nil	N	0.08	0	0	0	0
274	812328	S	59	M	2019065041	11.8	10500	50	41	8	1	169000	0.88	99.72	78	0.6	0.11	37	34	N	50	0.12	403	0.12	N	nil	N	0.08	0	1	0	0
275	813849	J	50	M	2019065632	12.2	4300	34	54	10	2	37000	0.90	105.56	76	0.32	0.1	194	122	PE Rt	66	0.04	420	0.12	N	nil	N	0.08	0	0	0	0
276	811579	R	51	M	2019064713	10	7200	75	15	8	2	55000	0.90	105.76	77	0.6	0.5	63	56	b/l infil	92	0.04	408	0.2	N	1mm in v4-6	tall T in v2-v4	0.08	0	0	0	0
277	813164	SD	50	F	2019065361	9.6	12000	50	43	5	2	130000	0.89	81.18	68	1.35	0.9	123	189	N	98	0.04	422	0.12	N	nil	N	0.04	0	0	0	0
278	814562	S	58	M	2019065837	12.9	6600	36	50	10	4	110000	0.08	1224.31	86	0.42	0.1	49	51	N	71	0.08	412	0.08	N	nil	tall T in v2-v4	0.08	0	0	0	0
279	634128	R	45	F	2019065896	10.6	12100	77	15	8	0	117000	0.90	88.48	71	0.5	0.1	33	46	N	101	0.04	449	0.2	N	nil	N	0.04	0	0	0	0
280	814685	R	45	M	2019065963	10.2	4300	38	50	10	2	72000	1.02	100.90	78	1.1	0.5	145	144	N	99	0.08	411	0.16	N	nil	N	0.04	0	0	0	0
281	814573	RK	31	M	2019065891	12.7	3600	64	20	10	6	110000	0.66	169.74	74	0.8	0.3	93	45	N	53	0.12	374	0.2	N	nil	N	0.08	0	1	0	0

Sr No	MRD	LV Sytolic Dysfunction	Pericardial Effusion	Valvular Defect	LV diastolic dysfunction	RWMA	LVEF	MISC	PAP	CKMB in IU/L	TR T in ng/mL	D DIMER ng/mL	MYO ng/mL	LDH U/L	Cardiac Manifestation	At the time of Discharge													14 days outcome				Co-morbidities
																HR	Pri	QRS	QTc	BBB	ST Elev	ST/T changes	Tpe	Ectopics	Brady arrhthymias	Tachy	Cardiac Manifestation AT THE TIME OF discharge	ECG	ECHO	Mortality	Cardiac Manifestation after 14 days of discharge	
233	848035	N	N	N	P	N	55	0	N	5	0.09	1710	135	220	myo	97	0.08	445	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
234	843908	N	N	N	P	N	62	0	N	6	0	200	45	220	normal	96	0.08	378	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
235	846585	N	N	N	P	N	62	0	N	5	0.05	1130	403	350	myo, brady	63	0.08	385	0.2	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
236	809616	N	N	N	P	N	55	0	N	5	0.05	5500	500	400	myo	106	0.04	492	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
237	810658	N	N	N	P	N	62	0	N	6	0	1200	25	220	myo	73	0.06	517	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
238	810590	N	N	N	P	N	62	0	N	5	0.05	346	352	320	myo, tachy	108	0.08	432	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CKD
239	813082	N	N	N	P	N	62	0	N	5	0	100	25	220	normal	85	0.08	415	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
240	807257	N	N	N	P	N	62	0	N	5	0.05	2360	237	220	myo	81	0.08	511	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
241	808880	N	N	N	P	N	55	0	N	5	0.05	4780	162	400	myo	100	0.08	446	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	CKD
242	810651	N	N	N	P	N	62	0	N	5	0	180	30	220	normal	99	0.08	429	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
243	811494	N	N	N	P	N	62	0	N	6	0	190	35	220	myo	69	0.04	425	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
244	767657	N	N	N	P	N	62	0	N	5	0	200	40	220	normal	75	0.08	433	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
245	812361	N	N	N	P	N	57	0	N	8.6	0.05	709	420	400	myo, brady	62	0.08	364	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
246	797689	N	N	N	P	N	62	0	N	5	0	80	40	220	normal	67	0.08	437	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
247	811186	N	N	N	P	N	62	0	N	5	0	70	30	220	myo	66	0.08	413	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
248	812233	N	N	N	P	N	62	0	N	5	0	60	35	220	myo	68	0.08	443	0.12	N	nil	N	iso	0	0	0	NORMAL	N	N	NIL	Normal	Nil
249	809747	N	N	N	P	N	62	0	N	5	0.1	4620	405	410	myo, tachy	105	0.04	388	AF	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	CKD
250	811746	N	N	N	P	N	62	0	N	6	0	80	25	220	normal	63	0.04	415	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
251	811491	N	N	N	P	N	62	0	N	6	0	80	25	220	normal	75	0.08	385	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
252	812212	N	N	N	P	N	62	0	N	5	0	80	25	170	normal	71	0.08	469	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
253	811563	N	N	N	P	N	62	0	N	5	0	80	25	170	normal	78	0.04	385	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
254	811618	N	N	N	P	N	60	0	N	7	0	80	25	180	normal	85	0.08	422	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
255	810611	N	N	N	P	N	62	0	N	7	0	80	25	170	normal	78	0.08	456	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
256	811593	N	N	N	P	N	62	0	N	8	0	80	25	160	normal	65	0.04	427	0.16	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
257	814561	N	N	N	P	N	60	0	N	9	0	80	25	170	myo	61	0.04	328	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
258	813808	P	N	N	P	global	28	0	N	5	0.07	2610	41.8	310	myo	86	0.04	442	0.12	N	nil	N	0.08	0	0	0	NORMAL	EF	N	NIL	Normal	Nil
259	815317	N	N	N	P	N	55	0	N	5	0.05	2370	170	300	myo	62	0.04	396	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
260	814677	N	N	N	P	N	58	0	N	5	0	100	30	170	myo	61	0.08	425	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
261	813854	N	N	N	P	N	59	0	N	11	0	100	30	170	normal	96	0.08	425	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
262	813766	N	N	N	P	N	62	0	N	14	0	100	25	170	normal	97	0.08	488	0.08	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
263	813888	N	N	N	P	N	60	0	N	5	0	100	30	170	normal	76	0.04	398	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
264	815404	N	N	N	P	N	61	0	N	5	0	100	30	170	normal	88	0.08	432	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
265	815439	N	N	N	P	N	66	0	N	5	0	100	25	170	normal	76	0.08	440	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
266	815243	N	N	N	P	N	55	0	N	6	0	100	30	160	normal	64	0.08	403	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
267	311952	N	N	N	P	N	56	0	N	5	0	100	40	170	myo	95	0.08	416	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
268	815275	N	N	N	P	N	57	0	N	5	0	100	30	150	normal	97	0.08	433	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
269	815224	N	N	N	P	N	58	0	N	7	0	100	25	170	normal	63	0.04	358	0.2	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
270	814913	N	N	N	P	N	57	0	N	7	0	100	30	150	normal	72	0.04	442	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
271	817054	N	N	N	P	N	58	0	N	5	0	100	60	170	normal	96	0.04	435	0.12	N	nil	N	0.12	0	0	0	NORMAL	N	N	NIL	Normal	Nil
272	815286	N	N	N	P	N	59	0	N	8	0	100	30	140	normal	87	0.04	424	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
273	614527	N	N	N	P	N	60	0	N	9	0	100	30	170	normal	99	0.04	412	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
274	812328	N	N	N	P	N	60	0	N	5	0	100	45	170	brady	63	0.12	403	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
275	813849	N	N	N	P	N	61	0	N	5	0	100	30	170	normal	66	0.04	420	0.12	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
276	811579	N	N	N	P	N	60	0	N	5	0.5	5000	205	370	myo	92	0.04	408	0.2	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
277	813164	N	N	N	P	N	62	0	N	5	0	160	50	180	normal	98	0.04	422	0.12	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
278	814562	N	N	N	P	N	63	0	N	12	0	170	60	170	myo	71	0.08	412	0.08	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil
279	634128	N	N	N	P	N	55	0	N	11	0	200	55	150	normal	100	0.04	449	0.2	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
280	814685	N	N	N	P	N	55	0	N	5	0	80	25	140	normal	99	0.08	411	0.16	N	nil	N	0.04	0	0	0	NORMAL	N	N	NIL	Normal	Nil
281	814573	N	N	N	P	N	56	0	N	15	0	90	30	170	brady	72	0.12	374	0.2	N	nil	N	0.08	0	0	0	NORMAL	N	N	NIL	Normal	Nil

Baba Farid University
of Health Sciences
Sadiq Road
Faridkot-151 203 (Pb). INDIA.
Phone : 01639-256232,256236
Tele/Fax : 01639-256235
Email – Examination@bfuhs.ac.in

No. BFUHS/2K19p-TH/10394 Date 9/9/19

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Aditi Mathur**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Spectrum of cardiac manifestations and their impact on the outcome of patients with dengue fever

Supervisor
Dr. Sandeep Chhabra
Assoc. Prof.,
Dept. of Medicine,
DMCH, Ludhiana.

Co-Supervisor
Dr. N. P. Jain
Prof.,
Dept. of Medicine,
DMCH, Ludhiana.

Dr. Bhupinder Singh
Asst. Prof.,
Dept. of Cardiology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

S P Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

IN

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

OCTOBER, 2018

Dr. Aditi Mathur

DEPARTMENT OF MEDICINE

DAYANAND MEDICAL COLLEGE & HOSPITAL

LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER
For the degree of	M.D. (Medicine)
Name of the candidate	Dr. Aditi Mathur
Supervisor	Dr. Sandeep Chhabra, Associate Professor, Department of Medicine, DMCH, Ludhiana
Co-supervisor(s)	Dr. N P Jain, Professor, Department of Medicine, DMCH, Ludhiana Dr. Bhupinder Singh, Assistant professor, Department of Cardiology, DMCH Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Dengue fever can cause a spectrum of cardiac manifestations which have an important bearing on prognosis. Early diagnosis and initiation of proper treatment can prevent significant morbidity and mortality.

Aim of Study:

1. To study the spectrum of cardiac manifestations in patients of dengue fever
2. To ascertain the impact of cardiac manifestations on patient outcome.

Materials & methods: This study will be performed in Dayanand Medical College and Hospital (DMCH) over a period of one year.

All cases of dengue fever with cardiac complications will be included in the study. Detailed history and physical examination findings will be noted as per proforma

Clinical Significance: A proper understanding of the cardiac manifestations can significantly improve patient outcome.

Keywords: Dengue, fever, cardiac complications.

(Signature) Candidate	(Signature) Co-supervisor	(Signature) Co-supervisor	(Signature) Supervisor	(Signature) Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis entitled **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** exist in the Department of Medicine, Dayanand Medical College & Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

Supervisor

Dr. Sandeep Chhabra
Associate Professor
Department of Medicine
DMCH, Ludhiana

Co-Supervisor(s)

Dr. N P Jain
Professor
Department of Medicine
DMCH, Ludhiana

Dr. Bhupinder Singh
Assistant Professor
Department of Cardiology
DMCH, Ludhiana

Dr. Dinesh Gupta
Professor & Head
Department of Medicine
DMCH, Ludhiana

Place : Ludhiana

Date : 18/12/18

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Aditi Mathur
Department	Medicine
Topic of Thesis	SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER
Likely date of appearing in PG examination	JUNE 2021
Date of enrollment	MAY, 2018
Name of Head of Department	Dr. Dinesh Gupta
1) Supervisor **2) Co-Supervisor(s)**	Dr. Sandeep Chhabra Dr. N P Jain Dr. Bhupinder Singh
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. & Gynae.
	8. Legal Expert
	9. Secretary, IEC
	10. Dean Academics
	11. Vice Principal
	12. Principal

Approved: Yes / No

Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	ADITI MATHUR 250, PHULKIAN ENCLAVE, PATIALA.
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	MD MEDICINE
4	DATE OF ADMISSION OF COURSE	MAY, 2018
5	TITLE OF THE TOPIC	SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH TROPICAL FEVERS – A PROSPECTIVE STUDY

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Dengue is one of the most important emerging infectious diseases in the world. It spreads by the bite of infected Aedes mosquito, that bites during daytime. As the disease is vector transmitted its highest incidence is seen during and after the monsoon in India during the months of July to November. The dengue virus belongs to the genus Flavivirus and has four distinct serotypes. The disease is reported in more than 100 countries; however, the burden of disease is particularly high in south east Asian countries. The disease causes considerable morbidity and mortality and requires intensive unit (ICU) care like mechanical ventilation, vasopressor support, blood or blood component therapy due to single or multiorgan failure.

Dengue is included in the list of tropical fevers and according to Moolani et al a considerable component of the burden of cardiovascular diseases is caused by neglected tropical diseases. [1] Dengue fever is an important cause of significant morbidity and mortality in India and has shown an increasing incidence with every passing year. In 2012 WHO declared dengue as "the most important mosquito borne viral disease of the world".[2]

Dengue fever presents as varied clinical spectrum of dengue fever (DF), dengue hemorrhagic fever (DHF), dengue shock syndrome (DSS), and expanded dengue syndrome (EDS). After the end of incubation period there are three phases of this disease- febrile, critical and recovery phase. Myocardial involvement occurs in expanded dengue syndrome.

The warning signs of dengue are: -

- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleeding
- Lethargy or restlessness
- Liver enlargement by >2cm
- Increase in haematocrit concurrent with rapid decrease in platelet count

The criteria for severe dengue are: -

- Severe plasma leakage leading to shock (Dengue shock syndrome) or fluid accumulation with respiratory distress
- Severe bleeding
- Severe organ involvement of: -

1) Liver: Increased level of serum aminotransferases of 1000 U/L or more.
2) CNS: impaired consciousness
3) Involvement of heart or other organs.

The exact mechanism of cardiac injury in dengue is not known, however it is postulated that there is direct invasion of the myocardial cells by the virus which causes inflammatory damage to the myocytes. The various inflammatory markers which are released by activated T cells cause suppression of myocardial contractility and disturbances in the conduction pathway. This leads to various types of conduction block and arrythmias.

The various cardiac manifestations include

- Sinus tachycardia

- Sinus bradycardia
- Nonspecific ST-T changes
- SA node dysfunction
- AV dissociation with variable degrees of heart block
- Tachyarrhythmias like atrial fibrillation and ventricular tachycardia
- Pericardial effusion
- Wall hypokinesia
- Elevation of cardiac enzymes

In severe form of the disease there is a higher incidence of cardiac abnormalities. [3] As the cardiac insult is transient no specific guidelines exist for its management. The treatment is to provide symptomatic support and maintain hemodynamic stability. The role of anti arrhythmics is not clearly defined. Atrial fibrillation is the most commonly clinically encountered arrhythmia, although it is rare.

Role of drugs like calcium channel blockers and beta-blocker in management of Atrial fibrillation (AF) in dengue patients is controversial in view of impending hypotension. However, some of the studies have shown use of anti-arrhythmics for non-self-limiting AF. AV dissociation, premature ventricular complexes, wall hypokinesia, nonspecific ST– T changes are transient and subside within 4 – 8 weeks of follow up. However, such patients require strict monitoring and any event of hemodynamic instability should be managed aggressively.

Early diagnosis and prompt initiation of treatment of cardiac complications can ensure a favorable outcome. It is imperative therefore, to study the various

cardiac manifestations in tropical fever and to know their impact on patient outcome in terms of morbidity and mortality.

6.2 REVIEW OF LITERATURE

In 1997, Lauer et al demonstrated that serum level of cardiac troponin was a very sensitive predictor of myocardial injury due to myocarditis. It was even more sensitive than endomyocardial biopsy and myocardial injury may be present even in absence of histological signs of myocarditis.[4]

Veloso et al in 2003 reported a case of 62-year-old man with dengue hemorrhagic fever who developed acute atrial fibrillation with rapid ventricular rate. The patient did not have any structural heart disease. The patient responded well to intravenous amiodarone. [5]

In 2007 Gulati and Maheshwari described atypical manifestations of dengue such as encephalitis, myocarditis, hepatitis and cholecystitis. In the cardiac manifestations they described myocarditis, conduction abnormalities and pericarditis. According to the authors most of the cardiac complications were asymptomatic and had a benign self-limiting course. [6]

Wichmann et al in 2009 studied cardiac involvement in dengue fever and noted that Sri Lanka experienced a dramatic increase in dengue cases (15,400) in the 2004 - 2005 season. They carried out a prospective study to investigate cardiac involvement in dengue virus infected patients during the 2004 - 2005 season in Peradeniya, Central Province, Sri Lanka. Cardiac involvement was defined as elevated levels of myoglobin, creatine kinase-muscle brain-type, N-terminal pro-brain natriuretic peptide, heart-type fatty acid-binding protein and troponin T. Twenty-five percent of dengue virus infected patients had one or more of the above tests with abnormal results in their study. [7]

Lee et al in 2010 reported a case of 65-year-old woman with dengue

hemorrhagic fever due to dengue serotype 3. The patient developed acute myocarditis and pulmonary edema, clinically masquerading as acute myocardial infarction as there was depression of ST segment on ECG and raised troponin 1 level. However, the patient recovered with supportive treatment in 3 days. [8]

Gupta et al in 2010 did a retrospective study on 28 patients of dengue hemorrhagic fever to detect cardiac involvement. They concluded that a significant number (71%) of patients of dengue developed asymptomatic involvement of heart as evidenced by raised cardiac enzymes (CPK-MB, S. troponin. T, LDH and SGOT). Myocardial involvement was subclinical as 2D-Echo was normal in 23patients (82%). The cause of raised cardiac enzymes in these patients was subclinical myocarditis. [9]

Miranda et al in 2013 evaluated cardiac involvement during dengue infection by using sensitive and specific biomarkers and cardiac magnetic resonance imaging which is considered to be the gold standard for evaluation of myocarditis and pericarditis. Cardiac involvement was observed in 15% of patients requiring hospitalization, with clinical manifestation ranging from mild elevation of cardiac biomarkers to myocarditis and/or pericarditis and death.[10]

Tarique et al in 2013 concluded that cardiac involvement may occur in dengue infection with ECG changes in both symptomatic and asymptomatic patients. Commonly noted finding were ST depression and bradycardia. [11]

Mohammed et al in 2015 reported a case of 24 year old male who presented to emergency department with history of sudden onset of chest pain, with prior history of fever with myalgia, joints pain since 5 days. The patient was found to be in shock and ECG revealed ventricular tachycardia [VT]. Post DC

shock with 200 joules, VT reverted and ECG showed sinus rhythm. Further investigation supported that patient was suffering from dengue myocarditis. [12]

This study by Sheetal and Jacob was done in 2016 to study the prevalence of cardiac manifestations of dengue fever and to find out the correlation of cardiac manifestations to the warning signs of dengue and severe dengue fever. The most common cardiac manifestations noted were transient rhythm abnormalities, of which sinus bradycardia was the commonest. There was no evidence of myocarditis in any of the patients. There was statistically significant correlation between cardiac manifestations and all the warning signs except persistent vomiting. Among severe dengue fever, fluid accumulation causing respiratory distress was found to have a significant correlation with the cardiac manifestations. [13]

Li et al in 2016 studied the characterization of myocarditis during the worst outbreak of dengue infection in China and concluded that myocarditis is a common complication of severe dengue infection. According to the authors the data about prevalence and characterization of myocarditis in dengue is still lacking. They studied 1782 cases of dengue and diagnosed 201 cases with myocarditis with a prevalence rate of 11.28%. Incidence of cardiac complications increased with severity of the disease. However, the prognosis of dengue patients with and without myocarditis remained the same. [14]

Francis studied cardiac involvement in dengue patients in 2017. He reported a wide variation in the incidence of cardiac involvement in dengue due to the difference in the criteria used to identify cardiac involvement. Most common cardiac rhythm abnormality noted was sinus bradycardia, which was often self-limited. Fulminant and fatal myocarditis was documented in a few cases;

though in general,

most cases with cardiac manifestations improved with supportive care. Only about one tenth of patients with biomarker elevation had evidence of depressed left ventricular systolic function on echocardiography. Higher incidence of depressed left ventricular ejection fraction was noted in dengue shock syndrome.[15]

Mukhopadhyay et al in 2017 studied 50 patients of dengue fever and noted rhythm disturbances in 8% of patients. AV block was the most common rhythm disturbance observed in 66.67% of patients. Cardiac manifestations in the form of ECG and echocardiographic changes were observed in 52% of patients with a positive correlation with the severity of dengue fever as defined by WHO criteria. Cardiac manifestations were also found to be significantly related to involvement of other organs. [16]

Nenavath et al studied the clinical profile and cardiac involvement in dengue patients in 2017.The results and follow-up of their study revealed that myocardial involvement of dengue infections run a benign course without long-term complications. The shock syndrome in severe dengue infections is most likely due to hypovolaemia and internal fluid extravasation. Dengue myocarditis was exclusively asymptomatic with no long-term sequelae. Two-dimensional echocardiography was the only reliable tool of investigation. Early diagnosis, strict monitoring and prompt supportive management reduced the mortality rate in dengue. [17]

Yadav et al in 2017 studied cardiac manifestations in patients of dengue and determined the correlation between these with the warning signs of dengue. The most common cardiac manifestations noted by the authors were transient

rhythm abnormalities, of which sinus bradycardia was the commonest. There was no evidence of myocarditis in any of the patients. There was statistically significant correlation between cardiac manifestations and all the warning signs except lethargy/restlessness and hepatomegaly. [18]

Chopra in 2018 studied 200 cases of dengue fever in Vadodra, Gujrat, India. The study was done to find out the prevalence of cardiac involvement in dengue patients and to find out the correlation of cardiac manifestations to warning signs and severe dengue hemorrhagic fever/dengue shock syndrome. The most common cardiac manifestation noted was transient rhythm abnormalities of which sinus bradycardia was seen in 66 (33%) patients. 45 (22.5%) patients had AV block and 71 patients (35.5%) had dengue hemorrhagic fever/ dengue shock syndrome of which 47 (35.5%) patients had myocarditis.

It was concluded that patients with dengue fever were at high risk of developing myocarditis and rhythm disturbance and therefore required a close monitoring. [19]

Sumana et al in 2018 studied the association of dengue fever and bradycardia. The clinical sign of relative bradycardia is a characteristic feature of dengue fever. They studied 60 patients of serologically positive dengue fever and observed that 55% had relative bradycardia and ECG showed sinus bradycardia in 38.3% of patients. Hence bradycardia as a clinical sign can help in early recognition of dengue complications and reduce mortality.[20]

6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To study the spectrum of cardiac manifestations in dengue fever.
2. To ascertain the impact of cardiac manifestations on patient outcome.

7. MATERIALS AND METHODS

This study will be performed in Dayanand Medical College and Hospital (DMCH) over a period of one year. All cases of dengue fever with cardiac complications will be included in the study. Patients above 18 years of age will be included in the study. In every case, a detailed clinical history and examination, with special attention to cardiac manifestations, will be taken. Diagnosis of dengue fever will be established by serological examination.

A specially designed proforma will be used to collect data on all patients. Written informed consent will be obtained from all patients before enrolling them in this study. SPSS software will be used for data analysis.

7.1 Source of Data

Data will be sourced from the indoor patients admitted in DMCH.

7.2 METHOD OF COLLECTION OF DATA

Patient's history, clinical examination and all investigations will be noted down as per detailed proforma.

INCLUSION CRITERIA

1. All patients of dengue fever with cardiac manifestations, of age more than 18 years, admitted in DMCH will be included in the study.
2. Only those patients will be included in the study whose diagnosis of dengue fever is confirmed by serological (ELISA) examination.

EXCLUSION CRITERIA

1. Patients with pre-existing cardiac manifestations will be excluded on the basis of history.
2. Patients who have fever due to causes other than dengue fever will also be excluded.

OUTCOME

Impact of dengue fever with cardiac manifestation on patient outcome will be assessed after 14 days in terms of mortatility, sudden cardiac death, Syncope, NYHA class of Palpitation, Dyspnoea, Chest pain, persistant ECG changes, LV dysfunction on ECHO.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate.All statistical calculations will done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

This study does not require any intervention to be conducted on the patient. Only those investigations are required for the study which are essential for diagnosis and clinical management of the patient.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached along with

LIST OF REFERENCES

1. Moolani Y, Bukhman G, Hotez PJ. Neglected tropical diseases as hidden causes of cardiovascular disease. PLoS Negl Trop Dis. 2012;6(6):e1499.
2. World Health Organization (WHO) Global Strategy for Dengue Prevention and Control, 2012 – 2020, Geneva: WHO Press; 2012. Available at : http://www.who.int/immunization/sage/meetings/2013/april/5_Dengue_SAGE_Apr2013_Global_Strategy.pdf
3. Arora M, Patil RS. Cardiac Manifestation in Dengue Fever. J Assoc Physicians India. 2016;64(7):40-4.
4. Lauer B, Niederau C, Kuhl U, Schannwell M, Pauschinger M, Strauer BE et al. Cardiac troponin T in patients with clinically suspected myocarditis. J Am Coll Cardiol. 1997;30(5):1354-9.
5. Veloso HH, Ferreira JA, de Paiva JMB, Honorio JF, Belki NCJ, de Paola AAV. Acute atrial fibrillation during dengue hemorrhagic fever. Braz J Infect Dis. 2003;7(6):418-22.
6. Gulati S, Maheshwari A. Atypical Manifestations of Dengue. Trop Med Int Health. 2007;12(9):1087-95.
7. Wichmann D, Kularatne S, Ehrhardt S, Wijesinghe S, Brattig NW, Abel W et al. Cardiac involvement in dengue virus infections during the 2004/2005 dengue fever season in Sri Lanka. Southeast Asian J Trop Med Public Health. 2009;40(4):727-30

8. Lee IK, Lee WH, Liu JW, Yang KD. Acute myocarditis in dengue hemorrhagic fever: a case report and review of cardiac complications in dengue-affected patients. Int J Infect Dis 14. 2010: e919–922.

9. Gupta VK, Gadpayle AK. Subclinical cardiac involvement in dengue haemorrhagic fever. JIACM 2010;11(2):107-11.

10. Miranda CH, Borges MC, Matsuno AK, Vilar FC, Gali LG, Volpe GJ et al. Evaluation of cardiac involvement during dengue viral infection. Clin Infect Dis. 2013;57(6):812-9.

11. Tarique S, Murtaza G, Asif S, Qureshi IH. ECG manifestations in Dengue Infection. Annals. 2013;19(4):282-85

12. Mohammed MZ, Venugopal K, Jaligidad K, Bharathraj MY, Reddy A. Ventricular Tachycardia in Dengue Fever. IJPCR. 2015;7(2):162-3.

13. Sheetal S, Jacob E. A Study on the Cardiac Manifestations of Dengue. J Assoc Physicians India. 2016;64(5):30-4.

14. Li Y, Hu Z, Huang Y, Li J, Hong W, Qin Z et al. Characterization of the myocarditis during the worst outbreak of dengue infection in China Med. J. 2016;95:4051-6

15. Francis J. Myocarditis in dengue fever. BMH Med J. 2017;4(3):77-80

16. Mukhopadhyay A, Kumar R, Singh BK. A study on cardiac manifestations of dengue fever. JMSCR. 2017;5(4):20736-45

17. Nenavath K, Valluri KK. Study of Dengue Fever, its clinical profile and cardiac involvement among paediatric population. IOSR-JDMS. 2017;16(8):60-62

18. Yadav RK, Kumar S. To study cardiac manifestations in patients presenting with dengue infection and to find out the correlation of

cardiac manifestations to warning signs of dengue. Int J Adv Med. 2017;4(2):323-8

19. Chopra NK. Study of cardiac involvement in 200 cases of Dengue fever in Shanti Infectious Diseases Clinic Vadodra, Gujrat, India. J Prev Infect Control. 2018;4:35

20. Sumana MN, Begum TM, Gowdappa BH. Association of Dengue fever and Bradykardia-The Established Fact, an evaluation. IJSR. 2018;7:39-41.

PROFORMA

NAME: AGE: GENDER:

MRD:

ADMISSION NO: TELEPHONE NO:

HISTORY:

- CHIEF COMPLAINT
- PAST HISTORY OF CARDIAC ILLNESS

VITALS:HR

RR

BP

TEMP

GENERAL PHYSICAL EXAMINATION:

Pallor	Icterus	Cyanosis
Clubbing	Lymphadenopathy	Edema

CLINICAL EXAMINATION:

CNS

CVS

Respiratory System

Per Abdomen

<u>HAEMOGRAM:</u>

- HAEMOGLOBIN:
- TLC:
- DLC:
- PLT:

Signature of Candidate **Signature of Supervisor**

CREATININE CLEARENCE:

LFT:

- BILIRUBIN:TOTAL:
- BILIRUBIN:DIRECT:
- SGOT:
- SGPT:

CXR:

- CARDIOMEGALY
- PROMINENT BRONCHOVASCULAR MARKINGS
- PLEURAL EFFUSION
- PULMONARY INFILTRATE

ECG:

- HR
- PRi
- QRS DURATION
- QTc
- Tpe
- BBB(RBBB, LBBB, NON-SPECIFIC INTERVENTRICULAR CONDUCTION DEFECT)
- ST ELEVATION
- ST/T WAVE CHANGES
- ECTOPICS (ABSENT, APC, VPC)
- BRADY ARRYTHMIA (ABSENT, SINUS BRADY,1 ST DEGREE HEART BLOCK,2 ND DEGREE HEART BLOCK, COMPLETE HEART BLOCK)

Signature of Candidate **Signature of Supervisor**

- TACHY ARRYTHMIA (ABSENT, AF, VT, VF, TdP)
- MISCELLANEOUS

ECHO:

- LV IDD
- LVIDS
- LVEF
- MR(ABSENT=MILD, PRESENT=MODERATE/SEVERE)
- TR (ABSENT, PRESENT)
- PAP
- PERICARDIAL EFFUSION
- E/A RATIO
- REGIONAL WALL MOTION ABNORMALITY

SOB PROFILE:

- CKMB
- TR T
- D DIMER
- MYO
- LDH
- IN HOSPITAL OUTCOME:(MORTALITY)

AT DISCHARGE:

ECG:

- HR
- PRi

Signature of Candidate **Signature of Supervisor**

- QRS DURATION
- QTc
- BBB(RBBB, LBBB, NON-SPECIFIC INTERVENTRICULAR CONDUCTION DEFECT)
- ST ELEVATION
- ST/T WAVE CHANGES
- ECTOPICS (ABSENT, APC, VPC)
- BRADY ARRYTHMIA (ABSENT, SINUS BRADY,1 ST DEGREE HEART BLOCK,2 ND DEGREE HEART BLOCK, COMPLETE HEART BLOCK)
- TACHY ARRYTHMIA (ABSENT, AF, VT, VF, TdP)

14 DAYS OUTCOME:(ECG,ECHO: LV DYSFUNCTION, MORTALITY, NYHA CLASS OF PALPITATION, DYSPNOEA, CHEST PAIN, SYNCOPE, SCD)

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....

am willing to participate in the study **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____
गवाह के हस्ताक्षर
नाम:

_____
रोगी के हस्ताक्षर/अंगूठे का निशान
नाम:

_____
अन्वेषक के हस्ताक्षर

_____
विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"SPECTRUM OF CARDIAC MANIFESTATIONS AND THEIR IMPACT ON THE OUTCOME OF PATIENTS WITH DENGUE FEVER" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ